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Original Articles and Clinical Cases.

HUGHLINGS JACKSON AND THE CORTICAL MOTOR CENTRES IN THE LIGHT OF PHYSIOLOGICAL RESEARCH.

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GENTLEMEN,—I am well aware that Horace of old has sometimes been accused of vanity for his ode "Exegi monumentum ære perennius." The poetical form, however, of the thought that pervaded the ode was perhaps only the expression of an antique simplicity; for the prophetic words of the same ode, "non omnis moriar," point to the fulfilment of a wish that pervades the breast of almost everyone. Nevertheless this hope of an existence reaching beyond what is in the more narrow sense mortal, has assumed a very different shape among various nations and religions and again among various professions and persons. One of the happy thoughts in this direction and one that first took a practical form in this country, aimed at preserving vividly and imperishably the memory of the mental achievements by which important men in our department elevated themselves above the great mass. The idea is to secure at stated intervals lectures upon some division of the science to which those men had devoted their best strength. Such a custom is in fact far more lasting than lifeless bronze and far better suited to the recognition of mental work.

We live in Time and it is therefore not vouchsafed to us to create anything absolutely imperishable. No one can

say whether or not the intellectual monument which this society intends to erect to Dr. Hughlings Jackson—the honoured man whom we still to our joy number as one of us—will be of such duration as the poet prophesied for his work: "As long as the Pontifex ascends to the Capitol with the silent virgin." If, however, it be permitted to judge by the development which this society has shown in the fourteen years of its existence, by the amount of intellectual labour that its members have performed, and by the influence that these labours have had upon the scientific views of the medical and philosophical world,—then this monument will have a long life, and so long as it endures, so long will it offer to the illustrious scholar the most worthy of all rewards: His thoughts will ever again rise from the seemingly lifeless dust, and will spur posterity on to renewed intellectual labour in the field that he cultivated.

If we examine carefully the most important efforts in the realm of the natural sciences, we find that two qualities have always been especially effective: keenness of observation and curbing the imagination. The first quality is far less an attribute of the senses than it is of association. Hundreds see the same event without its exciting any thought. One single person, however, sees this event and he sees it with other eyes, because it connects itself in his mind with numerous other conceptions, and forms with them a whole which the imagination at once portrays as completed truth. Precisely here, at the very side of this special pre-eminence of the inquiring mind, lies the danger. We learn the truth only when we stay upon the basis of reality, and when we prune back the luxurious shoots of the imagination, however they may for the moment please our inward eye. This is the ever renewed struggle *with* the hypothesis and *against* the hypothesis. It is not every inquirer that has been alike fortunate on every occasion in this struggle, as well as in general in the exercise of those two qualities. On the contrary we couple with almost every distinguished name the memory of some *one* particularly eminent scientific achievement. As for Dr. Hughlings Jackson there is no doubt that his merits are

of the most manifold character, and there is further no doubt that for English physicians his personality has not failed to be of influence upon the estimate made of his work. But in foreign countries we connect his name above all with the memory of one fact, namely, that he was the first one who referred the partial cortical convulsions to peculiar centres of innervation. Many another had already observed these convulsions, but he alone hit the right explanation for the phenomena, and, by referring the convulsions to such centres, he argued the existence of motor centres in the brain, and even told to a certain degree about what their position was, before physiological research was able with certainty to prove where they were. The scientific world has therefore, as you all know, given to these convulsions the name "Jackson's convulsions." Under these circumstances, whatever part of Dr. Jackson's work later lecturers on this foundation may choose as the point of departure for their observations, it may well seem justifiable, nay imperative for me, the first one called upon for an estimate of his labours, to choose the subject:

What development, in consequence of physiological researches, has the doctrine of the cortical motor centres experienced since those investigations of Dr. Jackson's.

I think that in this way I shall meet the wishes of this society. For when it summoned me to be the first of these lecturers, it doubtless did so for no other reason than because I was precisely the first one, now some thirty and more years ago, to confirm by *physiological experiment* and to define more closely that which Hughlings Jackson had concluded from *clinical* facts. I hope and I believe that the members present, in consideration of my connection with these researches, will deal leniently with all such defects of this lecture as may arise from my imperfect command of the English language.

The character of the task set me requires that its solution be sought in the main by way of history. As a necessary consequence, part of this lecture will offer to some of the members only what is well known to them, and will certainly offer to many members at least much that is

familiar. Still it may perhaps not be uninteresting to consider all these facts in a brief summary and to glance at them from the point of view from which they present themselves to a man who has for a long time occupied himself with them and with the questions associated with them. The material has in the course of these thirty years assumed very extraordinary proportions, and regard to time and to the occasion compels me to limit myself to those facts and to that part of the consideration of the facts which may be necessary to a clear, even if sketch-like, portrayal of the picture which we have to reproduce. I shall therefore be compelled to pass over numerous valuable contributions of English and other authors, even in the field of physiology, and to leave altogether to my successors in this office the consideration of the anatomical and pathological sides of these questions. In spite of this limitation it will at once be apparent that it is quite impossible to confine our view merely to the centrifugal functions of the cortical motor centres, and that we must needs, although only within certain bounds, busy ourselves also with the sensory functions of the brain.

The theme that is set before us points of itself to the proper beginning for our discussion. We have first to present the opinions which *Hughlings Jackson* uttered before the publication of the first physiological work on the cortical motor centres. Happily we have an article of his, termed "Study of Convulsions," in which he summed up these views of his immediately before the appearance of that work in the year 1870.

It will be best to proceed from Dr. Jackson's conception of the qualities of the brain substance. In reference to it he utters first of all with full decision the opinion that the gray matter and it alone has the property of starting motions, and in particular convulsions, a sentence that has ever been and ever remains for me an axiom. It is further his opinion that paralysis is produced by lesion of nerve fibres, a sentence the correctness of which, in this exclusive form, is open to objection. The genesis of motion from the gray matter is mediated by the contractions of the

arterial vessels, and that in such a way that every antecedent utterance of activity on the part of a certain district of the gray matter excites by means of the arterial innervation the activity of another district. We may then consider these processes as discharges which spread themselves out in the gray matter according to the manner of the distribution of its blood-vessels. And if we accept this conception, then on the one hand the processes in breathing, in walking, and in thinking will be in principle the same, and on the other hand we shall not need to grant a qualitative difference but only a quantitative one between the violent and irregular motions of the cerebral convulsions and the co-ordinated motions of normal life which are effected at a small expense of power.[1]

We have thus defined these motions as a united whole and have found that they depend upon the presence of the gray matter. It follows from this that the relative amount of the gray matter favours the production both of normal and of convulsive motions. Therefore the corpus striatum, in so far as it contains a less amount of gray matter, will be less easily, and the cortex, in so far as it contains a greater amount of gray matter, will be more easily, qualified to produce all these phenomena of motion. Indeed, Jackson presupposes that the latter are "to blame in severe convulsions at all events."

The relation of the cerebrum to the corpus striatum is to be conceived of in such a way that in the latter the more simple and in the former the more intricate co-ordinations of impressions and motions in time and space are represented. In the former numerous other elements come into play, whereas at every point of the brain cortex the units again appear which are present in the corpus striatum. Experience teaches that the motor functions of a whole half of the body can be annulled by the destruction of a

[1] This course of thought occurs often in numerous articles of Jacksons, for example, "Localisation of Movements," 1875, p. 33: "The inference is irresistible that there must be a motor as well as a sensory element in the 'organ of mind' which is faintly discharged when we 'think' of an object." "Slight discharges arise in the case of reproduction, strong discharges in the case of first production of sensible perception." "In the case of convulsions the discharge is excessive."

small portion of the corpus striatum, whereas a far greater destruction of cortex does not lead to paralysis. Now this is based precisely upon that growth of new formations of nerve substance, by which those aggregates of the corpus striatum are in the cortex pressed apart over broad surfaces. We must not, however, picture to ourselves the structure of the nervous system as if in one convolution only the motions of the arm, in another only the motions of speech, and in a third only the motions of the leg were represented. On the contrary, each separate convolution contains a multitude of elements of one unit of the corpus striatum, for example, of the arm, and a smaller number of elements of another of those units, for example, of the leg and of the face.

Taken as a whole the cerebral cortex is conceived of as an organ in which *motions and sensations* are represented, and in which there exists a definite kind of localisation for these motions and sensations, something after the manner in which Luciani conceived of them in one of his later works.

It is true that we learn but little here touching the part that the impressions on the lower and on the higher senses play in the cerebrum. Hughlings Jackson assumes that there is a certain segmentary connection of motor and sensory fibres with the corpus striatum, in so far as muscle nerves and the sensory fibres from the skin which covers these muscles meet there. He thinks that in this way it is possible to explain the interruption of the convulsion by the ligature of a limb. But he does not think that these bundles of fibres go as far as the cortex. As for the higher senses, we learn even, "that mere destruction of large parts of the hemisphere does not cause blindness, nor even defect of sight."[1]

[1] On this point we find in "The Localisation of Movements," p. 41, the following statements: "I wish to point out that I have suggested that disease of the posterior lobes, chiefly of the right one, although it does not produce loss of sight, does produce loss or defect of visual perception (imperception); or as I now prefer to say, defect of recognition." (Who is not reminded by this of Munk's soul-blindness?) Further, in a note at the same place: "I make four statements (1) Destruction of no part of the cerebral hemisphere produces loss of sight. (2) Discharges of parts of it (I think posterior part chiefly) produce coloured vision. (3) Tumour in it leads to changes in the optic nerve (optic neuritis), in consequence of which defect or loss of sight may or may not follow. (4) Disease of the posterior lobe (right) produces defect of perception."

If we then consider the way in which Hughlings Jackson speaks of the motor functions of the cortex as endowed with the qualities that we have described, the conviction at once forces itself upon us that in all his considerations and conclusions he was impeded and misled by the firmly rooted error of that day, one based upon the doctrine of Flourens, namely, that the actual motor centres lay only and exclusively in the corpus striatum. We can follow him and see how Hughlings Jackson's own experiences and conclusions constantly kept forcing him back to the cortex, and yet at the same time that he was fettered to the corpus striatum, because the universal opinion of research referred the cases of unilateral convulsions, which depended upon the destruction of routes of conduction that lay in the capsula interna, to centres that were supposed to lie in the gray matter of these ganglia.[1]

The next consequence of this conception of Hughlings Jackson's, in connection with his hypothesis as to the part played by the blood-vessels, is that he looks for the place from which the convulsions that he treats of issue, in the field that is fed by the arteria fossæ Sylvii, seeing that this artery supplies the corpus striatum and many neighbouring convolutions with blood. The very important utterances about the clinical symptoms that accompany aphasia agree with this conception; for an attack of convulsions that brings in its course a temporary loss of speech, always begins on the right side in the face or in the tongue, and no such defect of speech occurs when a similar attack begins in the right hand or in the right foot. Dr. Jackson says decidedly that the only conclusion he can draw is that the illness, on which that loss of speech depends, sits chiefly in that part of the corpus striatum which includes Broca's convolution, and in the same way that the *hemiplegia* that appears after such attacks is dependent upon changes in this region. Hughlings Jackson likewise states as a result of his experience, that convulsive attacks which show

[1] Compare "Localisation of Movements," p. 2: "At that time [1868] however, I believed the corpus striatum to be the part discharged in *convulsions* beginning unilaterally, although then and several years before I believed the convolutions also to contain processes representing movements."

tumour symptoms and which begin in the hand depend upon a lesion of convolutions of the arteria fossæ Sylvii. The suggestion that the cause of a convulsive attack that begins in the foot is perhaps to be sought in the neighbourhood of the thalamus opticus is less fortunate.

We have already seen that the cortex played in fact essentially no other part in Hughlings Jackson's view at that time of the case lying before us, than the further central gray matter. It must be capable of producing and storing great quantities of material for discharges or for explosions. And it was then necessary that this material, either constantly or in single attacks, should be discharged through all the lower centres by the use of the motor tracts. General epileptic convulsions and the convulsions named after Dr. Jackson differ from each other only in the one particular, namely, that the latter arise from discharges in convolutions which lie farther from the motor tract, whereas in the common epileptic attacks the discharges are in convolutions nearer that region. The influence of the cortex upon movement only goes as far as that. Todd's epileptic hemiplegia in particular does not depend directly upon organic unsoundness; it is the result of overwork of the nerve fibres which lead from the discharging part to the convulsing muscles.[1]

It is well known that one of the most important services rendered to science by Dr. Jackson consists in the description of the way in which these partial attacks of convulsions of his develop. He says in this passage: "Fits which begin in the foot have a different march from those which begin in the hand. When a fit begins in the hand it goes *up* the arm and *down* the leg. Now patients who have fits beginning in the foot tell me that the spasm goes *up* the leg and *down* the arm."

[1] "Disease in the hemisphere never causes palsy limited to one side, unless it also involves or squeezes the motor tract. . . . It does not cause hemiplegia, that is, by *destroying* a large part of the hemisphere. . . . The palsy depends upon secondary changes near the coarse disease, or rather on the 'overwork' of nerve fibres by the excessive discharge on them which these secondary changes permit. . . . The assertion is, that hemiplegia does not result from the *lack* of the part of the hemisphere *destroyed* by the disease."

"The fit begins most often in the parts that suffer *most* in hemiplegia, and these are always the muscles which are most concerned in arbitrary, isolated motion."

There are few cases in which the fits begin elsewhere "than in the side of the face (usually in the cheek), in the hand or in the foot; they very rarely begin in the upper arm or in the calf. . . . Fits beginning in the hands are common, fits beginning in the cheek and tongue are less frequent, fits beginning in the foot are rare. . . . The fits which begin in the hand, begin usually in the index finger and thumb; fits which begin in the foot begin usually in the great toe."

If we consider the total result of these doctrines of Dr. Jackson, we shall easily recognise from our present point of view, in what places and in how many directions they still needed completion and correction. And at the same time we grasp the fact that on the one hand clinics, on the other hand physiology, and finally anatomy, while each has shown its own right to independent research, still are to such an extent dependent upon each other, that the full recognition of truth can only arise from the common application of the results of all these sciences.

It is probably scarcely possible to prove this more strikingly than by the influence which the very first physiological researches as to the qualities of the cortex exercised precisely upon the conclusions drawn from those clinic and anatomic observations, and especially upon the theories of Hughlings Jackson.

These experiments are so well known that I may waive a closer description of them and content myself merely with the emphasizing of a few points which are hereafter to be discussed.

It was proved by experimental excitation, using the galvanic and the faradic current, that the muscles of the extremities, of the neck, the trunk, and of those innervated by the facial nerve, could be set in motion from certain narrowly bounded places of the gyrus sigmoides and of a neighbouring convolution in the brain of the dog. It was further discovered that the removal of very small portions

of the surface of the cortex of the gyrus sigmoides produced a peculiar disturbance of the motions. And, finally, Dr. Jackson's theory touching the charging of the cortex and its discharge in local and in general fits of convulsions, found a practical confirmation by the method of the Faradic excitation. As a matter of fact the cortex, and that even by a short series of merely weak inductive shocks, can be so charged that even after the stimulus has ceased local fits of convulsions arise, which may spread further and develop into general fits. *The phenomena of such a charging* have been more thoroughly examined and graphically presented by François-Franck and Pitres. A charging of this kind can be effected from more distantly existent points of the cortex, and for this reason the electric examination of the cortex should never be attempted without the control with the galvanic current, which does not lead to a residual charging of the cortex, or should only be attempted with single inductive shocks.

The way in which this discharge spreads out was described in general lines by the first discoverers themselves. The later researches of Luciani,[1] Franck and Pitres,[2] Unverricht,[3] and many others, showed in agreement with them that the stimulus imparts itself from the originally excited point to the neighbouring cortical regions. In particular, Luciani first of all, and then Munk,[4] Bubnoff and Heidenhain,[5] Ziehen[6] and others, showed that by the removal of a locally limited portion of the cortex such a discharge could be influenced to such an extent that the motors represented in the parts removed no longer convulsed in the former manner. Chiefly by pursuing such artificially excited fits of convulsions on the brain cortex

[1] LUCIANI, "Sulla patogenesi dell' epilessia." *Rivista sperim. di freniatria*, 1878 and 1881.

[2] FRANCK and PITRES, "Encephal." *Dictionn. encyclop.* (Dechambre).

[3] UNVERRICHT, "Experimentelle und klinische Untersuchungen über die Epilepsie," *Arch. f. Psychiatrie*, Bd. xi.

[4] MUNK, *Lehrbuch der Gehirnkrankheiten von Wernicke*, 1881, Band i., S. 240.

[5] BUBNOFF and HEIDENHAIN, "Ueber Erregungs und Hemmungsvorgänge innerhalb der motorischen Rindencentren," *Pflüger's Arch.*, Bd. xxvi.

[6] ZIEHEN, "Ueber die Krämpfe infolge elektrischer Reizung der Grosshirnrinde," *Archiv. für Psych.*, Bd. xiii.

of apes and comparing them with the monospastic and hemispastic fits, which were now better understood, it was discovered that all these facts agreed with each other and with the exemplary clinical description given of them by Hughlings Jackson. This comparison showed, however, at the same time, that the hypothesis set up by him was only correct in so far as it referred to the existence of cortical centres in general, but not in so far as concerned the assumption of manifold centres by means of which the different courses of these fits of convulsions were to be explained.

Little, however, as we could enter into the particulars of the researches which we first mentioned, just as little can we go into the details of the extremely numerous researches by means of which the facts we have just mentioned were rounded out. My task is rather to discuss the various theories according to which the newly-found facts were conceived and explained.

We may divide the questions here raised into two unequal groups, namely: (1) *The question whether the sum total of our experience justifies the assumption of centres in the brain cortex, and* (2) *the question which has received such extremely different answers, in what way such centres may be supposed to act.*

It would, of course, be the right thing to define at the very outset of this discussion, what is to be understood by the notion "centre," and all the more right because this expression has been applied in the literature in the most different ways and has given rise to disagreeable misunderstandings. But it will soon appear that no such universally valid definition can be given in a few words. Nevertheless we may content ourselves for the needs of the moment by understanding that a centre is a locality in the brain cortex, not bounded by lines, within which certain groups of conceptions are anatomically and functionally localised. In the case before us it would then be a matter of ideas of motion and of motor functions without any respect to the way in which we suppose them to have originated. The essential point is therefore the validity of the localisation,

or in other words the question as to whether the doctrine of Flourens with reference to mobility be still tenable or not.

The existence of such centres has always been and still is contested by Goltz and a number of his pupils, by Schiff and others. These opponents raised a series of objections to the force of the evidence of the experiments both in stimulus and in paralysis.

As for the former, Goltz[1] especially, among many others, has renewedly declared that they proved nothing at all in the face of the possibility of the diffusion of the electrical current. But he has never taken up my challenge to show me the place at which these currents could with any real effect diffuse, and he has finally given us to understand that he probably means the layer of fibres lying directly under the cortex. Moreover, the question was discussed with the greatest caution even in my first article. At that time, arguing on the one hand from the direct cortical excitation, and on the other hand from the excitation of the white matter by isolated needles, I had concluded that only the excitability of the latter had been directly proved, but that we could indirectly conclude at least the function if not the excitability of the gray matter in so far as under the circumstances we were of necessity forced to assume that those excitable fibres had their origin in the neighbouring cortical cells.

Since that time, however, the direct proof of the electrical excitability of the cortex, of the existence of centres in it, and the direct confirmatory proof against the objections raised, has been brought out in the most manifold way. It was found that after the undercutting of the cortex the electric stimulus only took effect in case an extraordinarily strong current was applied, but that neither the cutting around a centre, which did not hinder the diffusion of the current, nor its isolation by means of glass plates, which did hinder the diffusion, changed in any way the effects of the electrical stimulus, provided only that the

[1] Friedrich Goltz, "Ueber die Verrichtungen des Grosshirns," *Gesammelte Abhandlungen*, Bonn, 1881.

direct cortico-muscular path remained uninjured. And the experiments of Bubnoff and Heidenhain,[1] and as well those of François-Franck and Pitres,[2] were absolutely decisive; they proved that the gray and the white matter had a totally different kind of electrical reaction. The time of reaction, the time between the entrance of the current and the beginning of the twitching, is materially shorter in the case of excitation of the white matter than in that of the gray matter. The muscle curves, drawn in each, show the most essential differences from each other; and finally the white matter does not possess the power of being charged by inductive currents and of then discharging itself in local or general convulsions. A similar testimony is to be found in the different reaction of the cortex and of the marrow upon a number of poisons.

The interesting experiments of Horsley and Gotch[3] proved further that a negative variation (Du Bois-Reymond) of the current of the resting nerve in the spinal cord occurred when the corresponding cortex centre was excited, and only when precisely this centre was excited. Finally, within the last few months Larionow's[4] experiments have taught us that this negative variation can also be traced in the reverse direction, in so far, namely, that the currents of cortical action experience a locally limited change when peripheric stimulus is applied.

If we put all these facts together there is not room for the least doubt that even from the results of the different methods of electrical experiment alone, the existence of functionally different centres, especially of such as are of a motor character, in the brain cortex itself, would be made sure even if the results of the experiments in extirpation did not speak in the same sense.

[1] Bubnoff and Heidenhain, "Ueber Erregungs und Hemmungsvorgänge innerhalb der motorischen Rindencentren," *Pflüger's Arch.*, Bd. xxvi.

[2] Franck and Pitres, "Encephale," *Dictionn. encyclop.* (Dechambre).

[3] Horsley and Gotch, "On the Mammalian Nervous System," &c., *Philosoph. Trans.*, vol. clxxxii., 1891.

[4] Larionow: "Ueber galvanometrische Messungen der Ströme in der Rinde der Schläfenwindungen bei Reizung der peripheren Gehörorgane," *Monatsschr. f. Psychiatrie*, &c., Bd. v., 1889, S. 464. A more detailed publication than that in the passage cited, will perhaps dissipate many a doubt as to the author's results. I have occupied myself with similar experiments since the year 1874.

It is true that Goltz, whom we may doubtless regard as the representative of the opponents of the doctrine of localisation, has until of late contested this doctrine most zealously by his experiments in extirpation. We shall, however, see that his own experiments, in spite of all he may say against it, have really, nevertheless, although by a very roundabout way, led him back in the main to the very same mode of viewing the case that formed as long ago as the year 1870 the point of departure for my consideration of the functions of the cerebrum. I have always valued and recognised in the highest degree Goltz's services in the field of the physiology of the central nervous system. If he has in spite of that given occasion to unpleasant conflicts and thereby also to the spreading of great confusion over the questions with which we are busied, it is perfectly clear, for every one acquainted with the literature, that the reason for this lies in the unsuitable method of operation which he used at the beginning of his experiments. The moment that he applied methods which permitted, even if it was only an imperfect localisation, he reached results which it is quite possible to harmonise with mine and with those of scholars who think as I do.

It is well known that Goltz began his extirpations by flushing the matter of the brain with a jet of water which he brought to bear on the brain by means of variously shaped pipes after trepanning in one or more places. In this way he flushed the brain in the most different directions and in different depths, without telling what was the extent of the injury caused, and without, in my opinion, being able to control that extent. He himself alleges that by this process, by a pressure on the oblongata, in the case of very many animals there occurred a sudden cessation of breathing and of the pulse. In the case of other animals he mangled the brain with an instrument proposed by R. Ewald, a so-called scissors-borer.

If we wish to make experiments in *localisation*, it is —however much Goltz may have said or may say against it—a simple demand of logic that we either bring stimulus to bear or cause lesion *locally*, and that we then compare

with each other the phenomena produced in this way at the different places of the surface or of the interior of the brain. The methods that Goltz at that time applied were, however, clearly not suited to investigate the qualities of fixed localities of the cerebrum, in so far as these methods purposely injured large parts of it and extended unintentionally their effects far beyond the cerebrum, even as far as the medulla oblongata. Even though it is beyond doubt that in this way many a valuable bit of knowledge about the functions of the brain was attainable, no one can be surprised that Goltz at first came to the conclusion that not the *place* of the loss of substance, but only its *amount*, had a decisive influence upon the character of the disturbances. That was so clear to him that he even says in so many words: "In the subsequent statements I may therefore as a rule omit a sufficiently exact designation of the place of the lesions."

But the moment that Goltz applies methods, which, although still far from meeting the demands made by me, nevertheless no longer attack the whole cerebrum, but rather either its anterior or its posterior half, he finds, certainly to the surprise of his readers, that he is compelled to make, word for word, the following remark: "I read in different authors with the greatest astonishment the observation that I had declared that the matter of the brain is everywhere of like value."[1] When then Goltz in his sixth article also repeats the sentence, that he holds the assumption of small limited centres as more nonsensical than ever, he is still forced in the same article to concede that as a consequence of mutilation of the *anterior* half of the cerebrum, all motions become coarse and awkward, while in another passage he concedes that the dog operated upon *behind* moves all the muscles of his body with normal skill, has undisturbed sensation of touch, does not step into the air, and shows no increase of reflex excitability, but that on the other hand it suffers chiefly, though not exclusively, from disturbance of vision. Had Goltz taken a still further

[1] Goltz, "Ueber die Verrichtungen des Grosshirns, Fünfte Abhandlung," *Pflüger's Archiv.*, Band xxxiv.

step in the limitation of his operative lesions, he would surely also have seen a further limitation of the consequences of his operations, that is, a clearer localisation. I may refer here to my lately published experiments,[1] which prove the exceedingly great sensibility of the brain even towards apparently indifferent operations. It has become clear thereby that the mere uncovering of the brain to a slight extent, without lesion of its matter, may lead to evident disturbances of mobility, of sensibility, and of the power of vision. How can anyone then be surprised that the most manifold disturbances of functions should appear, if he opens the skull to a large extent and makes the greatest inroads upon the brain? Thus far Goltz has constantly evaded my challenge to compare the effects of the strictly localised lesions with each other. Another reason for misunderstandings I shall mention later.

Luciani's[2] doctrine stands in very close relation to these explanations. This scholar, whom I highly respect, reached, as is well known on the basis of his experiments, the view that the single centres overlap each other, and that in consequence, although every function can be injured the most from a certain central region, at the same time a number of other functions are also in a greater or less degree injured with it. This agrees with the above-mentioned view of Hughlings Jackson that in every gyrus a larger quantity of the one function and a smaller quantity of various other functions are represented. A peculiarity of Luciani's doctrine is the construction of a "centre of centres," which he places in the neighbourhood of what I call the centre of the facial nerve. At that point he thinks that all the rest of the centres, the one overlapping the other, are interwoven with each other, so that by an injury to this central centre the most severe psychical disturbances and the most complicated results are produced. Luciani ascribes a very special importance to this fact.

Nevertheless his whole doctrine, and in particular the

[1] E. Hitzig. "Ueber das corticale Scheu des Hundes," *Archiv. f. Psych.*, Band xxxiii., Heft 3.

[2] Luciani and Seppilli, *Die Functions—Localisation auf der Grosshirnrinde*, 1886; and "On the Sensorial Localisations," &c., Brain, July, 1884.

last-mentioned fact, is incorrect. It is, indeed, incontestable and *a priori* necessary that the single cortical centres be connected with each other, so, for example, that every sensory centre must certainly be combined functionally with motor elements of consciousness. But this connection does not exist in the way that Luciani assumes and it cannot be proved by the method that he applied. For this purpose such large extirpations as he practised are useless. If the mere uncovering of a narrowly limited cortical field occasions clearly appreciable disturbances of this whole field, and even changes in its neighbourhood, it is plain that severe lesions in large cortical fields must much more certainly lead to manifold disturbances of the functions of the neighbourhood, and thus Luciani's results are to be explained. For the purpose that he had in view he should have applied both the method of the very least excitation and the method of the very least lesions. A single experiment of this kind proves the falsity of Luciani's whole doctrine. If, for example, we set a small trepanning bit at the place that corresponds to his centre of centres, and if we then cause a small or even a somewhat larger lesion at this point, but at the same time see to it that the wound heals aseptically, this operation remains, in very many cases, without visible influence upon the functions of the animal operated upon. Eckhard[1] reached, in this respect, the same results that I myself reached. Now a very much smaller lesion of the gyrus sigmoides produces severe disorder in the functions of the extremities and of the eye, therefore we should look for much more severe phenomena in the case of the lesion of so important an organ as the centre of centres. Luciani's error is simply to be explained by the fact that the so often mentioned locality lies about in the middle of the convexity of the brain, so that a coarse attack upon it is precisely best fitted to make all the rest of the convexity suffer with it.

If we draw the sum total of all these experiences, we find that the paralysing experiments speak in the same way as the excitation experiments, in other words, that all experi-

[1] ECKHART, C. "Das sogenannte Rindenfeld des Facialis in seiner Beziehung zu den Blinzbewegungen." *Centralblatt f. Phys.*, Bd. xii., Nr. 1.

ments, in so far as they are entered upon with the necessary precautions, prove the existence of centres in the cortex, and that in so far as they are entered upon without such precautions, they have produced no facts that would be inconsistent with the cortical localisation.

If we now turn to the second of the questions set before us—*the question as to the functions of these centres*—we must proceed from the description of the facts which their first discoverers were able to observe as a consequence of very small extirpations, and which, in the main, so far as the motor side is concerned, retain the same appearance in the case of large lesions.

These experiments showed that such lesions in the case of a dog never produce an actual paralysis in the form in which it appears in men, and in fact that, as later experiments showed, this was not even the case when the operation was on both sides and symmetrical. On the other hand, the extremities reached a condition which we defined as showing *that the animals operated upon had but a defective consciousness of the condition of their limbs, and that they had lost the power of forming for themselves complete conceptions as to these limbs.* This condition displayed itself chiefly in that the animals used their limbs actively in the most unsuitable way, and that they did not correct the most unsuitable positions which were passively given to their limbs.

These facts have been enlarged in the most manifold manner in the thirty years that have passed since that time. Inasmuch as the same experiments were also extended to other species of animals, the consequences of similar experiments differed more or less, doubtless, from the consequences shown by the experiment on the dog. But none of the further experiments have in the least changed any of the actual results that we reached then by the conduct of the dogs upon which we operated.

The enlargement of our knowledge has, however, led to a series of different theories as to the significance of those centres. The chief theories are: (1) The motor theory; and (2) the sensory theory.

The principal representative of the motor theory is D.

Ferrier,[1] a scholar to whom, as all know, the physiology of the brain owes, among other things, the discovery of a number of new facts in the realm of the excitation experiments. In the question now under consideration he proceeded from paralysis experiments, which he made in the motor region of apes. He reached therein, we use his own words, the following conclusions: "That the thus produced affection is a purely motor one, and the centres or regions in question are strictly motor, but motor in a special or limited sense, viz., psychomotor,"[2] and that "strictly cortical lesions of the motor area do not cause anæsthesia in any form, and it may be laid down as a rule, to which there are no exceptions, that if anæsthesia is found along with motor paralysis, the lesion is not limited to the motor zone."[3] "Though an animal does not react so readily to sensory stimulation of the paralysed side, it does not follow that this is due to diminished or absent perception of the stimulus. An animal may not react or react less energetically to a sensory stimulus, not because it does not feel it the less, but because it is unable, or less able, to do so from motor defect."[4]

Ferrier declares in this way most decidedly that sensory disturbances are lacking, while other authors, and among them especially Munk[5] and Mott,[6] and of late also Bechterew,[7] declare in just as decided a manner that they are present. Besides, even Ferrier has a short time ago in co-operation with Turner[8] in large experiments of extirpation found hemianæsthesia combined with hemiopia. But all these symptoms are explained by the latter author "by a dynamic influence exerted by the magnitude of the

[1] D. Ferrier, "The Functions of the Brain," second edition, 1886.
[2] *Ibid.*, p. 362.
[3] *Ibid.*, p. 378.
[4] *Ibid.*, pp. 374, 375.
[5] Munk, "Ueber die Functionen der Grosshirnrinde," 2 Aufl., 1890; and "Ueber die Fühlsphären der Grosshirnrinde." Sitzungsberichte der Akademie der Wissensch., 1892-1896.
[6] Frederick W. Mott, "The Sensory function of the Central Convolutions of the Cerebral Cortex," *Journal of Physiology*, vol. xv., 1893.
[7] Bechterew, "Ueber corticale Centren beim Affen, *Neurologisches Centralblatt*, 1898, p. 140.
[8] Ferrier and Turner, "An Experimental Research," &c., *Philos. Transactions*, vol. cxc. B., 1898, pp. 1-44.

lesion on the sensory centres or tracts which were not organically injured." "This explanation receives support from the fact that in smaller cerebral lesion—for example after the removal of the so-called 'leg area' alone—no such symptoms occurred."[1]

Finally, a very short time ago Schäfer[2] has come over altogether to Ferrier's side, in so far as he states that apes, after larger or smaller extirpations in the motor zone, still felt the slightest touch of a straw, &c. According to him "it cannot therefore be the case that the motor paralysis which is produced by a lesion of the Rolandic area is due to a sensory disturbance. And it also follows that tactile sensibility is not localised in the same part of the cortex from which voluntary motor impulses directly emanate."[3] We perceive that we have here astonishing differences of opinion as to observations of what is purely matter of fact, such differences as in other cases scarcely occur in reference to explanations.

My own experience as to the ape does not put me in a position to pass a judgment of my own in this question. I can only do this by way of conjecture, and by using the further literature as well as the experience I have had as to the dog and as to man. But even with reference to the dog, Ferrier speaks no less decidedly. He declares that the sensibility is not injured by lesions in the motor zone even of these animals, and says in so many words: "It is inconceivable that brains constructed on the same anatomical type, should differ fundamentally in their physiological organisation, and we may well assume that, however much the facts of experiment on the motor centres of dogs may appear opposed to those of monkeys, they are not so in reality![4]

If we remember one of those theses of Hughlings Jack-

[1] FERRIER and TURNER, "An Experimental Research," &c., *Philos. Transactions*, vol. cxc. B., 1898, p. 36.

[2] A. SCHÄFER, "On the Alleged Sensory Functions of the Motor Cortex Cerebri, *Journal of Physiology*, vol. xxii., No. 4, Nov. 25, 1898.

[3] *Ibid.*, vol. xxii., p. 312.

[4] D. FERRIER, "The Functions of the Brain," p. 361.

son, that the single elements of the subcortical centres in the cortex are pressed farther apart by the intrusion of other organisations, we can concede the last sentence, but, as we shall still see, without reaching the same conclusion as Ferrier.

It can under no circumstances be in any way doubted that any lesions at all in the motor part of the gyrus sigmoides of the dog are always and without exception combined with disturbances which affect the sensibility of the whole limb, that is to say, of the skin, of the muscles, of the joints, &c., and that these disturbances can in no case be an illusion caused by purely motor disturbances, or, more correctly expressed, by a "motor defect." In this reference to a matter of fact I agree with Schiff, Goltz, Munk, and the others, that is to say, with authors whose views I in other respects most decidedly oppose. If, then, Ferrier is of another opinion on this point, that can only be based upon the circumstance that he has not yet fully enough studied these "motor disturbances" of the dog.

Since the dog then undoubtedly shows disturbances of his sensory functions as a consequence of those lesions, it appears to me to be *a priori* impossible, or to speak with Ferrier, it would be "incomprehensible" if such disturbances were not to be observed in the ape. The reasons why they have escaped individual observers may be of a manifold description. They may, for example, depend upon the differences in the extent and depth of the lesion; this would agree with the fact that Ferrier and Turner found hemianæsthesia in the case of great extirpations, whereas Ferrier alone, in what appear to have been smaller extirpations, did not find hemianæsthesia. On the other hand, we cannot deny the correctness of Schäfer's conclusions, when this distinguished scholar, after he had found the sensibility intact in the palsied limb of the ape, says that the motor paralysis could not possibly be based on a totally imperceptible sensory disturbance. However, I shall have to return to this question in discussing Munk's doctrine.

The theory according to which the disturbances mentioned, and therefore also the normal motor actions, are to

be referred to disturbances of the sensory functions, has been very differently expressed by different authors.

The first one whom we have to name here is Schiff,[1] to whom, aside from the discovery of many other important facts, we owe the discovery of the cortical sensory disturbances. To put it briefly, in almost all his works Schiff considers the disturbances of motion that are to be observed in dogs to be first of all the consequence of a disturbed sensation in the skin, of a disturbed sense of touch, but is not willing to have either one symptom or the other referred to the lesion of cortical centres. On the contrary, he denies the existence of such centres.

We can dispose very quickly of the series of proofs which he bases upon his explanation of the effects of electrical stimulus. For in the complete edition of his essays, although he formally declares that he still holds to his old opinion, he simply omits the plan chosen to demonstrate his theory and all the arguments based upon it, after I had proved to him its internal impossibility.

Nevertheless, he has not had much better luck in his considerations as to the results of the paralysing experiments. Ferrier, as we have seen, regarded the cortical disturbances of motion as purely motor and rejected any and every concurrence of sensibility, whereas Schiff asserts precisely the opposite, namely that the destruction of these centres produces no motor paralysis at all, but merely destroys the sensibility to touch and *therewith the consciousness of the position and situation of the limbs.* Schiff has at every opportunity accused me of changing my view as to the motor nature of those disturbances of motion, and has claimed that our views are no longer very far apart. This last may to a certain degree be the case, but the first is totally incorrect, since I never spoke of a purely motor function of these centres in the sense that Schiff attributes to me. I have from the very beginning and without any cessation, as I have just represented it, designated these disturbances as disturbances of the perceptive activity. On the other hand Schiff has himself in the course of the years

[1] SCHIFF, *Gesammelte Abhandlungen.*

changed his opinion in the greatest measure conceivable. For he has at last not merely reached the position that I have held from the first, but he has also in reference to the ape and with the aid of a number of considerations, which are altogether my own, with all decision expressed the view that *motor paralysis* can be produced by lesion of those cortical centres.

Schiff did, it is true, originally concede the existence of such centres in general, but he repeated over again that they could not lie in the cortex but must lie down below, and he heaped up proof after proof of this. Later he allowed this question to fall altogether out of the discussion. Originally, as we have said, he was only willing to deduce the disturbances of motion from a disturbance of the sensibility of touch. Later, however, he refers them to disturbances of the consciousness of the limbs and of the external parts of the body, that is to say, he explains them precisely as I do, and even citing my own words. And finally, Schiff has himself expressed the opinion that, as we ascend in the series of animals, centres advance from the subcortical regions even into the hemispheres, and this might in particular well be true of the centre of locomotion. In connection with this, from the fact that an ape does not grasp a fig which it clearly wishes to seize, he concludes that a paralysis is present similar to that of the hemiplegic man. Therefore, in his own words, a centre that mediates motion—a moving centre—must be injured. And if he had injured it by his operation, it must then be present at the place operated upon.

A more complete conversion than the one depicted is scarcely possible, for it goes very much beyond the measure that we observed when we treated of the successive works of Goltz.

Munk for his part assumed at the outset a totally different position. Whereas Schiff referred the disturbances of motion that were observed exclusively to the lesion of sensory ways in the cerebrum, and denied almost to the end the existence of cortical centres, Munk[1] on the contrary stood up from the

[1] MUNK, *Ueber die Functionen der Grosshirnrinde*, 2. Aufl.

beginning for the strictest localisation of the cortical functions and that to a degree which in certain respects goes far beyond my own views. On the other hand, he also refers the motor disturbances now under consideration to lesions in the sensory sphere, it is true more in appearance than in fact. I say "more in appearance," because he designated the parts that I called *partly* excitable[1] *partly* motor[2] region, by the name "independent spheres of feeling."

As a matter of fact Munk himself in his first communications named this section of the cortex a "motor sphere," and it was not until his third communication in the year 1878 that he used the name "sphere of feeling," supposing at the same time that he could thereby lead to a deeper understanding of this section of the cortex. He thought that he could compass this by localising all the feelings and the conceptions of feeling of each part of the body, that is to say of the skin, of the muscles, and the innervation feeling and conceptions of feeling, excluding, however, the common sensibility in the respective part of the sphere of feeling.[3] According to this we have to do in the sphere of feeling only with perceptions and conceptions which flow from the sensations of feelings, and therefore the conceptions of motion *alone* in the sphere of feeling are the causes of the so-called voluntary motions.[4] Correspondingly, lesions of these spheres produce according to their size the temporary or permanent loss of a part of, or of all, feeling and conceptions of feeling of the part of the body in question—psychical paralysis, or cortical paralysis of that part of the body.

[1] A remark is here necessary against Ferrier. On page 225 of the second edition of his work, "The Functions of the Brain," he has thought it necessary to observe, against my word "excitable," that there is no reason to assume that any parts of the cortex are not excitable. This observation is a stroke into the air, for he will find that on page 19 of my book, "Untersuchungen über das Gehirn," in the footnote, I have defined the expression "unerregbar" (not excitable) in the following words: "We call here, without any prejudgment, all those parts 'not excitable' from which we can produce no twitchings."

[2] As for the expression "motor," I have repeatedly defined what I wish to be understood by this word, which I use for the sake of brevity. On page 65 of the work cited in the foregoing note, I protested, among other things, expressly against Mr. Ferrier's statement which makes me say that I had produced paralysis by experiments in extirpation.

[3] MUNK, *Ueber die Functionen der Grosshirnrinde*, 2. Aufl., p. 32 and pp. 38, 39.

[4] *Ibid.*, 2. Aufl., p. 48.

First of all we must contest some of this doctrine as not correct, or, at any rate, not proved. Even if it be correct—and that follows clearly enough from Schiff's experiments—that the *conceptions of feeling* are injured by lesions in these spheres, I see no proof advanced for the statement that this also holds good for *feeling*, at least so far as the cortex is directly concerned. Indeed, it is for *a priori* and experimental reasons very probable that all feeling is formed subcortically and that only its apperception as well as its endowing with local signs takes place in the cortex, and we must not forget that the emphasizing of the relations of the local signs to the cortex is one of Munk's services to our science. In so far then as an injury of feeling itself occurs, it is probably to be referred to a checking of the functions of the subcortical organs. Munk was finally compelled to concede this, although at first only in the sense that even the sensations of pain from the extremities arise in the opposite extremity regions,[1] whereas he had originally excluded their participation in those apperceptions. In so far then as the dog is concerned, there is at present not the least reason to doubt the doctrine *that in the extremity regions only the conscious working over of all the feelings that arise subcortically to more or less well localised conceptions of feeling takes place*. In the second place, it is not correct to say that all the parts of the sphere of feeling behave alike in the relations mentioned. Only a short time ago Eckhard[2] proved that "the cortex field of the orbicularis had no influence on the *reflex* activity of the subcortical centre for the motions of the eyelids."

And finally the theory as to the origin of the muscular motions from this "sphere of feeling" needs a limitation which we shall make in a moment; but, with this limitation, Munk's theory agrees so exactly with the theory that I myself suggested, that it is impossible to accede to his claim that he himself first showed the way to the due understanding of the functions of these regions. You will

[1] Munk, *Ueber die Fühlsphären der Grosshirnrinde*, Sitzungsber, I. Mitteilung, p. 31, 1892.

[2] Eckhard, C., "Das sogenannte Rindenfeld des Facialis in seiner Beziehung zu den Blinzbewegungen," *Centralblatt f. Phys.* Bd. xii., No. 1.

remember that I had defined in general the character of the disturbances mentioned " as disturbances of the consciousness of the extremities as well as of the power to form for oneself complete conceptions of them," and it will not have escaped you that in this general definition Munk's definition is contained so far as it is correct. Furthermore this definition leaves room for enlarging the latter at pleasure, an enlargement which I at a later date, and that with reference to certain disturbances that arise in case of lesion of the extremity region, disturbances in the influence of the objects of sight upon the movements of the animals, shaped as follows, namely, that in consequence of such lesions external circumstances—sometimes of the muscles (&c.), sometimes of the objects in space—are not taken into account by the sensorium for the movements of the limb affected, but only for these.[1] As for the rest I have in another essay touched upon the question—" whether the centre that is thus brought forward be the organ that alone rules the muscular movement in question, or whether there be still another motor organ within the same chain of action."[2] This question I answered in the sense that Munk later accepted, that I found the cause of the so-called voluntary movements not *only* in the conceptions of motion in the sphere of feeling, for this is not correct, but that I also beheld in them *one* of these causes and a necessary component of these motions. In fact the conceptions of motions *in themselves* lead only in a limited range of cases to movements, and in all other cases there is need of the intervention of another excitement of the senses or of an intellectual occurrence in order to disengage the conception of movement and therewith the act of motion.

Before I pass on to the presentation of my own view as to the nature of the functions of those centres, you will permit me to remind you that a glance at what I have already said teaches: (*a*) that the scholars who have occupied themselves most particularly with the experimental

[1] Hitzig, " Untersuchungen über das Gehirn," Neue Folge, *Reichert's und Du Bois-Reymond's Archiv.*, 1876, H. 5, S. 705.
[2] *Untersuchungen über das Gehirn*, 1874, p. 60ff.

study of the brain, have finally, even if in part after long struggles, acknowledged, even if in different degrees, the differences of value in the cortex and therewith the localisation of the psychical functions in this organ;—(*b*) that all scholars without exception have come to the opinion that by lesion in what I call the motor zone the motions of the animals are impaired;—(*c*) but that there are differences of opinion as to whether only the motor functions or whether other functions also are injured, so that from this point again differences of opinion arise as to the reason for and the method of origin of the motor functions and of their disturbances; although these reasons are it is true sought by most authors, in agreement with the view expressed by me at the outset, in the conceptive activity or in a disturbance of the conceptive activity.

If we follow up the further reasons for the differences of opinion which still obtain, we shall find that they consist in the main in two circumstances:

(1) In the fact that the knowledge won by a number of scholars touching various animals and man has been applied without sufficient respect to the anatomical and physiological differences of the brains of these species.

(2) In the fact that functions, which were more or less lost after such lesion, really or apparently return.

I shall reach the exposition of my own views most simply if I try to dispose of these difficulties, so far as that is possible in the present state of our knowledge, and if I therein start out again from the brain of the dog, the functions of which are best known to me from my own experiments.

The disturbances in motion, which follow upon lesion of the gyrus sigmoides of this animal, may be divided into various groups. We are well acquainted with the disturbances which I myself first described and which in the main consist in the circumstance that the dog, actively, brings his extremities into *abnormal positions*, and that it, passively, allows them without opposition to be brought into such positions. I myself at that time referred these peculiar phenomena to the lack of the conceptions which

the normal dog forms of the condition of his organs of motion and especially of his muscles. Since then Charlton Bastian[1] has uttered a similar view, one however that, so far as the dog is concerned, I cannot accept entirely as my own. According to his view those motor centres are centres for the totality of *kinæsthetic impressions*. But we shall soon see that they possess a more broadly measured importance.

They certainly serve, however, also for the apperceptive working up of the kinæsthetic impressions, and it is not without value that we should sum up the totality of these impressions, as Charlton Bastian has done, under the name kinæsthesis. Lately I have, with reference to numerous works of others, tried to show that the totality of these impressions, that are supplied by the various apparatus that serve for orientation and locomotion, are wrought over to conceptions of a lower order touching the relation of the body and of its single parts to space, and that then the organ of consciousness, the cortex, deals with these conceptions of a lower order as with a given whole, without being able to pierce into its details. The place in which this last process, so far as it is possible to fix it, is effected, is according to the results of our researches the gyrus sigmoides, in man therefore a part of the Rolandic region. The determination of this method of viewing the case is of the more importance, because all the corticopetal, and probably, with certain modifications, also all the corticofugal processes proceed in a like manner.

The conceptions of feeling which we obtain from painless handling of the organs of the skin, do not directly belong to the kinæsthetic sensations, although they doubtless stand also in close relations to the motions. I already mentioned above that they also may be in a high degree and in part lastingly injured by lesions of the gyrus sigmoides. It is true that some, as for example Charlton Bastian, have denied that we can learn anything worthy of belief from animals about their conceptions of feeling. But I cannot

[1] CHARLTON BASTIAN, "The muscular sense its nature and cortical localisation," BRAIN, April, 1887.

agree with this view to its full extent. For we do learn sufficiently reliable points as to the internal processes in animals by means of motor reactions that follow with the regularity of law. We may, I concede, still remain in doubt upon the basis of the single experiment, as to whether these reactions take place under the concurrence of conceptions or not. Nevertheless comparative researches give us information about them with a probability that verges upon certainty. I learn then from the dog that has been operated upon, by the unilateral lack of the usual motor reactions, that the sum of his kinæsthetic and tactile impressions is no longer applied to the formation of conceptions for the member affected.

I have not spoken on this occasion of *pain-exciting impressions*, and I shall even now only say briefly that I regard the search for cortical centres for these—a search which has been chiefly practised by English scholars—as hopeless, for the very simple reason that dogs that had been robbed even of their whole cerebrum still showed to Goltz[1] upon the usual attacks the usual utterances of pain. According to this and according to the sum of the further experiments in this line, into which I cannot enter singly, *the feeling* of pain must, analogously to the kinæsthetic sensations, be formed *subcortically*. It is, however, in the highest degree probable that this feeling, in contrast to these sensations, is apperceived in broad sections of the brain cortex as a conception of pain, and that it is only in so far provided with local signs as the pain-exciting stimulus that touches the outer skin is accompanied by the touch stimulus which is accompanied by local signs.

In addition to the motor disturbances mentioned, there comes yet another group, out of which I myself described a number of symptoms as long ago as the year 1874.[2] The most important thing consisted in the fact that the dogs operated upon, as already referred to, with their affected forepaw blindly *step into the air* from the edge of the table, even when their power of sight is not destroyed. They behave

[1] Goltz, "Der Hund ohne Grosshirn," *Pflüger's Archiv.*, Bd. 51, 1892.
[2] Hitzig, "Untersuchungen über das Gehirn," Neue Folge, iv., *Reichert's und Du Bois-Reymond's Archiv.*, 1874, S. 440.

then, as I already at that time stated, as far as their fore-paw is concerned, as if for this member the power of sight did not exist, or as if the impressions of sight were not applied to forming conceptions for it. An observation of Goltz as to the tongue points to the same thing, although this scholar has never been willing to join me in my method of viewing the facts. It is, as is well known, not possible to grasp with the fingers a sound dog's tongue that is hanging out; but if we extirpate the corresponding centres it is easy to grasp it. Therefore here also visual conceptions of the dog are no longer applied for the motions of the tongue, which are as for the rest not impaired.

The third group of motor disturbances comprises such as present most or all the characteristics of paralysis as we so well know it in man, and which have been designated by the motion of the paralysis of isolated, intentional motion. I have already mentioned above the description Schiff gave of such a paralysis produced in the case of apes, in so far as such animals run and climb, but are not in a position purposely to grasp a fig with the paralysed hand and to put it into their mouth. Goltz was the first one to describe precisely the same condition for the dog, in so far as dogs, who have learned tricks, lose after the operation the power to give the paw. In the same way we must explain the conduct that I have described in the case of a dog that is hanging in the air, and who is not able to draw away by itself the paw that is hanging down stretched out and paralysed, when it is threatened with a sharp pin.

In all these cases we see that the further conceptions of the animal, the total of which we are accustomed to call his will, in whatever field of the senses they may have arisen, are no longer in a position to excite the motions that are necessary to the carrying out of the will, the moment that the centre I have named is extirpated for the fore-limb.

The relation of the gyrus sigmoides to the faculty of sight assumes a very peculiar position in these considerations. Goltz had originally asserted that the faculty of vision was affected just as much by lesions in the frontal as by lesions in the posterior lobe. Even if he did later, as already men-

tioned, modify this statement in so far as to declare that the power of sight was more severely and lastingly injured by lesions in the posterior than by lesions in the frontal lobe, that did not change the doctrine that the frontal lobe stood in direct relation to the power of sight. Munk indeed has constantly denied *any* such relation, since he referred the disturbances of vision that occurred in the case of such lesions to subsidiary lesions which were supposed to have affected his sphere of sight. Nothing can be more incorrect than that. I myself had long ago confirmed the simple fact, and the same confirmation has been offered by numerous other scholars, for example, Exner and Paneth and Luciani. Of late, however, I have established the fact that such disturbances of vision arise under circumstances that exclude every subsidiary lesion.

The question now arose whether the dog had only *one* cortical centre of vision in the occipital lobe, or whether he had still another in the motor sphere. This question seemed open to experimental solution on the basis of the following considerations:—If the dog, according to the latter assumption, really possessed two such centres, then the successive lesion of these two centres must produce a summing up of the disturbances of vision. But if we wait until the disturbance of sight that has occurred after a superficial lesion of the occipital lobe has disappeared and then follow it up by a superficial lesion of the gyrus sigmoides, there will not only be no summing up—that is, the vanished disturbance of vision does not reappear in an increased measure—but it will even not appear at all. It follows from this that the disturbance of vision which arises after lesions in the motor zone is only *indirectly* dependent upon this zone, *that the latter does not serve for vision*, and that in consequence the disturbance of vision can only be based upon a process of inhibition that is passed on into the centres of vision.

If anything at all can confirm the doctrine of the localisation of the functions of the brain and remove the last doubts that were conjured up by the Goltz experiments, it is this fact. The true nature of the motor centres appears

to be perfectly cleared up by it, and the ways are pointed out by means of which the individual fields of the brain cortex influence each other, as well as the manner of this influence.

If we wished to apply the knowledge thus gained from the *dog* to the *primates*, we should strictly speaking have to draw into consideration to a large extent the comparative physiology of motion. But I must waive that and limit myself to the consideration of the facts lying nearest at hand.

The most striking fact consists in any case in the circumstance that the exceedingly manifold disturbances in the psychical functions of the dog can be induced by an exceedingly slight lesion of the gyrus sigmoides, whereas far larger lesions in the motor zone of the ape give occasion to much less pronounced and complicated phenomena. We observe the same thing, and that in a much more pronounced measure, in the case of lesions which touch the Rolandic region in man. But if we descend in the series of animals, from the dog downwards, we find that the lower the animal stands in this series, the fewer and fewer portions of his motor system are represented in such a way in the brain cortex, that their existence would be provable by means of the electrical stimulus, and that the isolated intentional movement of these portions would be destroyed by the extirpation of the brain cortex. From this we may draw the general law, that on the one hand with the progressive development of the individual and of the brain constantly more and more central organs advance from the subcortical up into the cortical sphere and become subject to the consciousness, and that on the other hand these organs, as the consciousness developes itself more and more, are moved farther apart from each other by the fact that their separate elements differentiate themselves more and more and associate themselves perhaps with new formations which serve consciousness. If the disturbances of sensibility are lacking in the case of relatively large lesions in the Rolandic zone of the ape, and only occur just like temporary disturbances of vision in the case of very great extirpations,—if disturbances

of sensibility are wanting in 60% of the cortical lesions of this region in man,—and especially if what Wernicke calls "tactile paralysis" is only met with in comparatively rare cases,—all *this* points by no means to a *fundamental* difference in the organisation and in the physiological functions of the animal and human brain. On the contrary we can only conclude from this that the individual cortex fields must be extirpated to a particular extent and in a particular combination, if those peculiar disturbances are to be produced in the formation of the conceptions which proceed from the physiological changes in the organ of the skin and in the kinæsthetic organs. And further it is necessary so to arrange that extent and combination, as that the formation of these conceptions be not hindered by contemporaneous motor or intellectual paralysis.

I am very far from saying that the relation of sensibility to mobility or the representation of sensibility in the brain cortex of man is fully explained. On the contrary, I think that the most important tasks of the near future lie in this direction. But I also believe that the way in which these tasks are to be solved is designated by the course of thought that I have just sketched.

The other difficulty above mentioned, *The apparent restoration of functions that were apparently lost*, is to be solved by similar considerations.

We have known for a long while that animals of a lower order without a cerebrum or with only a rudimentary cerebrum are capable of very complicated forms of motion that are excited in a reflex way and that are mediated by *special lines of communication* with the intercentral and centrifugal course of which we are but imperfectly acquainted. In higher animals there is associated with this system a second motor system that stands in manifold relations to it and that serves for *conscious arbitrary motion*. This second system starts in the motor centres to which I have referred and takes its course in the pyramid tract, which constantly developes itself more and more with the higher development of the animal. If we extirpate these centres and these tracts it is true that conscious arbitrary

motion ceases in the accustomed forms and up to a certain degree in such animals and to a far higher degree in man. Nevertheless I had already said in my first treatise: "it is certain that in this case still further places and courses *must* stand open for some motor impulse or other in order that it may be born and may speed away to the muscles." Since then we have learned, by the experiments of Goltz and Munk, that what I then proved for the centres of the extremities also holds good for experiments with the removal of all motor centres. The experiments of E. A. Schäfer,[1] H. Munk[2] and others, have further taught in what way motor impulses from other than the motor regions of the brain cortex of the animal can become effectual: they have shown that conjugated eye and head motions occur upon excitation of the occipital lobe, that is to say motions that are to be paralleled with the *locomotor* associated motions of the animals. From this alone it was then to be concluded that such associated motions were not started merely by the motor region but also from large districts of the brain cortex.

Another series of experiments leads us to similar conclusions. Schiff[3] had proved that arbitrary motion was not destroyed by cutting through the pyramids. These experiments have with like success been prosecuted of late in a more precise way with manifold variations by Starlinger,[4] Redlich,[5] Prus,[6] H. E. Hering,[7] and others. A series of these experiments showed that the electrical excitation of the cortex, even after the cutting of these courses of communication, still called out epileptic-like and other motor

[1] E. A. Schäfer, "On Electrical Excitation of the Occipital Lobe," &c. *Proc. Royal Soc.*, 1888, vol. xliii. "Experim. on the Electrical Excitation, &c." Brain, 1888.

[2] H. Munk, "Sehsphäre und Augenbewegungen," Sitzungsberichte, 1890.

[3] Schiff, *Lehrbuch der Physiologie*, 1858, 1859, S. 306.

[4] Starlinger, "Die Durchschneidung beider Pyramiden beim Hunde," *Jahrb. f. Psych.*, Bd. xv., 1897.

[5] Redlich, "Beiträge zur Anatomie und Physiologie der motorischen Bahnen bei der Katze," *Monatsschr. f. Psych. u. Neurol.*, 1899, Bd. v.

[6] Prus, "Ueber die Leitungsbahnen und die Pathologie der Rindenepilepsie," *Wien. klin. Wochenschr.*, 1898.

[7] H. E. Hering, "Ueber Grosshirnrindenreizung nach Durchschneidung der Pyramiden oder anderer Teile des centralen Nervensystems mit besonderer Berücksichtigung der Rindenepilepsie," *Wiener klinische Wochenschr.* 1899, No. 33.

stimulatory effects, and they thus proved that the motor part of the cortex commands those subcortical centres and the courses of communication that radiate from them; these radiating courses probably continue, according to the researches of Redlich and of H. E. Hering, through the tegmentum of the crus cerebri, and according to the researches of Homén,[1] and especially of Redlich, in the spinal marrow, probably in the fasciculus marginalis anterior and in the fasciculus intermedio-lateralis.

That restoration is therefore no longer in the least surprising. If in the cortically maimed dog and in a certain sense also in the ape we at first observe a confused mixing up of, or even a total lack of, the motor expressions, that is to be explained, so far as *obstruction* or shock effects do not occur, by the fact that the remaining motor impulses are given off in an unaccustomed and irregular form; and then with the cessation of the shock effects there gradually arises an accommodation to the new conditions and at the same time a breaking up of the way upon still existing courses of communication. Munk has tried to follow up and explain in a detailed way that which I here call the "breaking up of the way," but I am not able to grant to his essay more than the value of a hypothesis.

If we now, according to the task set us, compare the standpoint of science to-day with the theories that Hughlings Jackson set up thirty years ago, we find, as must be expected from the defects attaching to human research, that some of his views have as a whole or in single parts needed to be corrected, while doubt to-day still reigns as to others, but that the most essential of them have found a brilliant confirmation at the hands of physiological research,—to say nothing of clinical and anatomical research. If we turn our eyes from that which will gradually pass into forgetfulness, we must in the first place emphasise, as an indelible leaf in the history of medicine, Jackson's doctrine of the cortical convulsions, both the general principle of the charging and of the discharge that makes inroads on the neighbouring

[1] HOMÉN, *Contribution expérimentale à la pathologie et à l'anatomie de la moelle épinière.*

domains, and the numerous details by which this principle was explained and firmly grounded; in it especially the part determining the muscles in which the cortical fit begins and the sequence in which they are affected by the fits after different beginnings.

In close connection with this stands the theory which sought the cortical motor centres in the region of the arteria fossæ Sylvii, and especially which brought the centre for the muscles that serve speech into local relations with the Broca convolution.

I am likewise convinced that his view of the cortical combination of motion and sensation has proved to be correct in principle. Both this view and the doctrine of the cortical pressing apart of subcortical unities by new combinations, will certainly in the future play a leading part in our views of mental occurrences, even though it may seem necessary to round out or to correct many a point in detail. The general relation also of the subcortical formations to the cerebral mechanism appears precisely, according to the latest researches, again more in the light in which Hughlings Jackson and the elder school viewed it, although the original argument for those views of Dr. Jackson's proceeded from an erroneous presupposition as to the part played by the internal capsule and the neighbouring gray matter. It may also to-day even still appear doubtful whether these gray masses are capable of an independent primary charging, or only of a secondary one through the cortex.

Taking it all in all we see, even in the part of Dr. Jackson's scientific work that I have described, the work of a man of whom any nation may rightly be proud.

The first physiological treatise in the field which we have entered upon to-day, remained for years unnoticed because of the thunder of the cannon which altered the fate of Central Europe. In the development of more peaceful times a literature that we can scarcely compass has attached itself to this treatise. We, too, we labourers in the realm of the natural sciences, to whatever nation we

belong, are soldiers and form an army, but an army that knows of no separation into different camps as a result of national or other interests, an army that is united in the one interest, in the search after truth, in the battle against ignorance and against all the hindrances which some men would like to oppose to our endeavour to reach our ideal aims. It may count as an expression of the mutual character of our cause that I, as a foreigner, was called to-day to do honour to a scholar of this country, and for this reason also I have discharged this flattering task with joy and pride.

THE CONDITION OF THE MEDULLATED FIBRES OF THE CORTEX CEREBRI IN TWENTY-FIVE CASES OF INSANITY.

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THE subject of the condition of the medullated fibres of the cortex cerebri in persons dying insane has not attracted much attention.

In 1884 Tuczek published his "Contribution to the Pathological Anatomy and Pathology of Dementia Paralytica," the research being concerned, as the title indicates, with a particular form of mental disorder. He found in all cases (seventeen) more or less considerable atrophy of the medullated fibres in certain areas of the cortex. In six control cases of other forms of mental disease, Tuczek got only negative results as regards nerve-fibre atrophy. Zacher, at a later date, examined for disease of the medullated fibres in various kinds of psychoses, and found that these were atrophied in other conditions as well as in paralytic dementia, thus modifying Tuczek's views. Zacher's work is the fullest with which I am acquainted, and I have found a comparison with his results very useful, the investigation being of an analogous nature with the present, though in methods of preparation of specimens and other points there are differences. Tuczek employed Exner's osmic-acid-glycerine method, but the well-known disadvantages attaching to this—such as lack of durability, swelling of sections, cost of osmic acid, the fact that small sections only can be

examined, and that in œdematous states of the brain some of the medullated fibres will not stain with osmium—have led to its abandonment in favour of the hæmatoxylin methods based on Weigert's process. A doubt may, however, be permitted as to whether these methods bring out the finest medullated fibres as well as in Exner's process.

Zacher worked with Friedmann's process, but the objection to this and the Friedmann-Flemming modification is, as he recognises, that the outermost zone is liable to be stained too deeply by the osmic acid employed, and thus the fibres there are obscured; further, the preparations are not uncommonly unsuccessful, for some unknown reason.

T. Kaes, whose writings upon the medullated fibres of the cortex are well known, has strongly recommended Wolters's method of staining the fibres. This is also known as the Kultschitzky-Wolters method, because the hæmatoxylin solution used is that recommended by the former (hæmatoxylin pur. cryst. 1·0, alc. abs. q. s. ad solv., acetic acid 2 per cent. sol. 100·0). Sections are left for twenty-four hours in the stain in the incubator. Kaes recommends that they be left for two to three days at 42° to 45°. He considers the method superior to Weigert's, and equal in results to that of Exner. It brings out the finest fibres. Kaes continues to speak well of the method in his later writings, and Passow, writing in 1898 upon the medullated fibres in certain gyri in sane persons, says that after four years' use he cannot speak too highly of the Wolters's method as modified by Kaes. This modification consists in the employment of Flemming's solution in place of Müller's for hardening. By this means Kaes finds that even better results are obtained; the tangential fibres especially are well brought out. Our experience is that an undesirable shrinkage of tissue occurs when Flemming's solution is used; also, that the outermost zone of the cortex is frequently obscured by a black deposit. But as regards the Kultschitzky-Wolters method we are quite able to endorse the experience of Kaes and Passow. We find that the fine fibres are very well brought out and that the durability of the stain is considerable. It is now two years

since this work was commenced, and a very large proportion of the sections show undiminished stain. The increased vascularity often present in morbid sections of cortex is well brought out.

In order to secure more certain results we have kept sections, as a matter of routine, in the stain for seven days in the cold, and thereafter for two days at a temperature of 38° to 40°. Brittleness has but seldom been a source of trouble.

Except in the earliest cases, sections were taken from the precentral region, central gyri, temporal and occipital regions. In the earliest cases the section did not comprise more than a single gyrus, or two gyri, with intervening sulcus; but in the later, and majority of cases, large sections were taken, embracing several gyri (up to thirteen), with intervening sulci. It is a possible objection to this plan that the gyri do not receive the individual care in the process of differentiation which would be given were each stained separately. But a certain measure of control in differentiation of the gyri is generally feasible with large sections. Occasionally it happens that a gyrus or two has to be left rather more "chromy" than one would desire, in order that the properly differentiated may be preserved; but this appears often to be due to disease in the gyrus. Everything considered, we are much in favour of the large sections, as giving a very instructive comparative representation of the gyri and their associated sulci. Most of our sections measure 4 to 6·5 cm. by 2 to 4 cm.

For the purpose of this investigation, following Edinger and others, the medullated fibres of the cortex are regarded as composed of the following:—(1) The zonal layer, containing the tangential fibres; (2) the superradiary layer; (3) the interradiary meshwork ("external association-fibres of Meynert"). Between (2) and (3) is placed the condensed layer of transversely-disposed fibres known as Baillarger's layer (or Gennari's, in the occipital region). Lastly, and apart from the above more or less transversely-disposed fibres—which are in this article, for brevity's sake, called "cross-fibres"—the projection-fibres or radii (4) have to be considered.

The origin and connections of the cross-fibres of the cortex—as far as present knowledge permits a statement—may briefly be referred to.

In the zonal area, which corresponds to the molecular or *external* layer of authors, the medullated fibres may be regarded as (*a*) prolongations of the axis-cylinders of the nerve-cells described as existing in that layer by Cajal, namely, the fusiform, triangular, and polygonal nerve-cells; the first two varieties are said to have several axis-cylinders; (*b*) prolongation of the axis-cylinders ascending to the molecular layer from their cells of origin in the pyramidal and polymorphous cell-layers; (*c*) the medullated fibres may also be regarded as partly composed of the terminals of the nerve-fibres, which can be followed into the white matter of the hemisphere, "the cells of origin of which are unknown" (van Gehuchten). The latter may, of course, be situated anywhere in the same or opposite hemisphere.

The superradiary layer and the interradiary meshwork correspond approximately to the layer of pyramidal cells (*middle* layer); the layer of small pyramidal cells answering approximately to the superradiary layer, that of the large pyramidal—including Betz's cells—to the interradiary meshwork.

Lastly, the layer of spindle or polymorphous cells (*internal* layer) is placed in the lowest part of the interradiary meshwork. The medullated fibres of the superradiary layer and the interradiary meshwork may be regarded as composed of (*a*) myelin-enveloped axis-cylinder processes of the cells of the middle and internal layers, which processes are ascending and descending, and of the collaterals of these (if we are justified in regarding these as clothed in myelin in part of their course); (*b*) the nerve-fibres above mentioned under (*c*) will also assist in forming the superradiary layer and interradiary meshwork. The paucity and delicacy of the superradiary fibres, in comparison with the interradiary, may be explained, perhaps, by the smaller size of the cells from which the former spring.

A reference to the order of development of the medullated fibres of the cortex will be opportune here, following

the description of Kaes. In the fœtus, between the fifth and sixth months, the cortex is free from medullated fibres. The first to penetrate are the projection-fibres, the fibræ propriæ of Meynert (between cortex and medulla) developing about the same time. Next come the fibres of the interradiary meshwork (Kaes's "external association fibres of Meynert"), and then, about the same time, the fibres of Baillarger's layer and of the zonal layer. Whilst the fibres of these three layers become gradually more numerous, the superradiary layer begins to show (Kaes's "fibre-band of the second and third layer"). The "primary deposit" is now complete. In this is developed the second system of fibres ("der in Gebrauch genommenen Faser"), thicker and more prominent than the primary, parallel or oblique in disposition. They appear first in the interradiary region, later in the superradiary, then in Baillarger's layer, finally spreading over the whole cortex "as the highest expression of development of the cortical fibres" (Kaes).

Twenty-five brains from cases of mental disorders were examined, embracing cases of mania and melancholia of various degrees, paralytic dementia, delusional insanity, imbecility, and terminal dementia.

In addition, a control specimen was examined from a man aged 46, who died suddenly, and in whom a fibroid state of the heart-muscle was found. The only objection to be brought against this case was the possibility of syphilis as the cause of the cardiac state. But however this may have been, the specimen furnished a large number of gyri which, in the light of other experience, were evidently normal. This control was supplemented by Kaes's description of the cortical medullated fibres in the various regions of the hemisphere in a normal brain. In addition, various of the cases furnished specimens of normal gyri, occurring in association with diseased ones, in the different regions of the hemisphere.

In summarising the condition of the medullated fibres the terms "slightly," "moderately," and "greatly" (or "extremely") degenerate have been used, according as the fibres exhibited the slighter or more aggravated forms of

degeneration, tabulated in the scheme according to which the work was done; though, of course, the classes pass into one another. (The condition of "scantiness" is also mentioned separately.)

Thus, with respect to the cross-fibres: if scanty, thin, short, faintly stained, the degeneration is put down as "slight." If, in addition, they are irregularly stained, broken, of uneven calibre, the fibres being mostly small, with large, thick fibres few or absent, the degeneration is "moderate." Whilst it is "great" if the fibres exhibit the above conditions in aggravation together with varicosity and tortuosity. In extreme cases the fibres are represented by rare dots and shreds, or may be absent.

With respect to the projection-fibres or radii: "slight" degeneration connotes the case in which the bundles are thin, with scanty fibres and faint staining, with the terminals of the radii thin, broken, badly stained, irregular in course. "Moderate" degeneration is where, in addition to the above, the bundles fall short, the fibres being broken and thin, unduly wavy in course and irregularly stained. And "great" degeneration connotes the above in aggravation, with uneven calibre of fibres, varicosity and tortuosity of terminals and also of trunks.

The long, thick, deeply-stained fibres met with in the different layers (especially the interradiary and the tangential) in normal specimens—which I take to be the fibres of Kaes's second system—are called, briefly, the "strong" fibres.

The medullated fibres running at right angles to the surface, and passing between the tangential fibres and the deeper parts of the cortex (and probably ascending from or descending to the white matter, in some instances) are called in this article "perpendicular" fibres.

The cases examined are as follows, summarised as much as appears desirable.

(1) H. E.—On admission: acute mania. Causation: influenza and trouble. Clinical course: remained maniacal for four months, then showed partial dementia; phthisis present on admission, progressed, and he improved somewhat in mind. At death: mental state was one of partial

dementia. Cause of death: phthisis. Total duration mental disorder: 6½ months. Age at death: 32.

Frontal gyri.—Tangential and superradiary fibres in fair number, and moderately degenerate. Interradiary meshwork: abundant, but fibres very thin. Radii: normal, excepting terminals, very degenerate. Vascularity + and general, with chrome about the vessels.

Summary.—Moderate degeneration of cross-fibres of outermost two layers; terminals of radii involved in the superradiary degeneration, and especially affected. Vascularity +.

Occipital gyri.—All cross layers appear normal, and radii also. No vascularity; no chrome. Strong and perpendicular fibres in fair number in the various layers.

Summary.—A normal specimen.

In this, the first specimen examined (acute mania passing into mild dementia), gyri were only taken from two regions. Fibre degeneration is moderate in the frontal region, and vascularity proportionately increased.

(2) W. J.—On admission: senile melancholia. Causation: heredity + senile changes. Clinical course: melancholia passing into dementia. At death: complete dementia. Cause of death: senile brain atrophy. Total duration: 4 years. Age at death: 75.

Frontal gyri.—Tangential fibres: exceedingly few and very degenerate. Superradiary: moderately degenerate and very scanty. Interradiary: moderately degenerate and scanty. The cross-fibres are rather worse on the sides. Radii: moderately degenerate, and terminals more so. Vascularity and chrome +, especially on sides.

Summary.—The medullated fibres diseased throughout, most so in the outermost part of cortex (terminals of radii sharing). Tangential about the worst. There are variations in the degree of degeneration in the different parts of the same gyrus (fibres in all layers more affected at one part than another). Maximum increase in vascularity and chrome deposit goes with the maximum fibre-degeneration.

Parietal gyri.—Tangential fibres: very scanty and very

degenerate; replaced by connective tissue in parts. Superradiary: rather scanty and moderately degenerated. Strong fibres absent in these two layers. Interradiary: normal, many strong fibres. Radii: trunks normal but terminals very degenerate. Chrome + and general. Vascularity +, but only in foci in outer parts.

Summary.—Cross-fibres degenerate in outer two layers, especially tangential. Terminals of radii share in degeneration. Vascularity is chiefly + with fibre-degeneration.

Occipital gyri.—Tangential fibres: very scanty and very degenerate; connective tissue and chrome deposit in outermost zone. Superradiary still more so. Interradiary: normal. Radii: ditto, terminals very degenerate. Some perpendicular fibres, but degenerate. Vascularity and chrome + and general.

Summary.—Fibres considerably diseased in outermost parts, including terminals of radii. Superradiary worst. Vascular changes not commensurate with those of nerve-fibres.

General summary.—In this case of senile melancholia with dementia the two outermost layers of cross-fibres and the terminals of the radii, which are situated at the level of the superradiary layer, show considerable disease in different parts of the cortex. The conditions are rather worse—the diseased fibres showing deeper in the cortex—in the frontal region. Vascularity and chrome increased, on the whole, in association with fibre-degeneration.

(3) C. W.—On admission: acute melancholia, with ideas of persecution also present. Causation: business troubles. Clinical course: melancholia throughout; attempted suicide. At death: melancholia. Cause of death: pleuro-pneumonia; cardio-arterial degeneration. Total duration: 2½ years. Age at death: 43.

Frontal gyri.—Tangential fibres; very scanty, very degenerate. Superradiary: scanty, very degenerate. Interradiary: extremely scanty, very degenerate. Radii: scanty and degenerate. A few perpendicular fibres, but degenerate. Specimens very vascular and chromy.

Summary.—All cross layers show much degeneration without important distinction, and this extends to radii. Vascularity and chrome + with fibre-changes.

Parietal gyri.—Tangential fibres: not scanty, very degenerate. Superradiary: ditto. Interradiary: normal. Radii: ditto. Vascularity and chrome +, but less than in last.

Summary.—Two outermost layers only affected, not scanty, but degenerate. Vascularity and chrome in accordance.

Occipital gyri.—Tangential fibres: very scanty and very degenerate. Superradiary: scanty, very degenerate. Interradiary: very scanty, slightly degenerate. Radii: very scanty and very degenerate. Vascularity and chrome much +.

Summary.—Gyri very degenerate, with vascularity and chrome in association.

Temporal gyri.—Tangential fibres: scanty, moderately degenerate. Superradiary: very scanty, moderately degenerate. Interradiary: scanty, moderately degenerate. Radii: scanty, slightly degenerate. Vascularity +, more than in last; chrome +.

Summary.—Cross-layers moderately degenerate; radii involved in less degree. Vascularity and chrome + in association.

General summary.—In this case of melancholia the medullated fibres generally show disease, the interradiary scarcely better than the two outermost layers, which are equally bad, advanced in the frontal and occipital regions about equally, least in the parietal. The radii sometimes equally involved, but on the whole less. Vascularity and chrome + in association with fibre-degeneration.

(4) J. E.—Admitted three times in three years with acute mania. Discharged recovered twice. On the third occasion remained more or less acutely maniacal for about fifteen months, when she lapsed into a state of chronic mania. This continued until death, weakness of mind becoming more apparent. Cause of death: myocarditis. Total duration: 15 years. Age at death: 65

Parietal gyri, "A."—Tangential fibres: fairly numerous, moderately degenerate. Superradiary: scanty, very degenerate; worse than tangential. Interradiary: numerous, slightly degenerate; good Baillarger layer. Radii: scanty, terminals moderately degenerate. No vascularity; chrome: slight. Perpendicular fibres in fair number, tortuous. Strong fibres in good number.

Parietal gyri, "B" (another part).—Tangential fibres: scanty, very degenerate. Superradiary: very scanty, very degenerate. Interradiary: ditto. Radii: scanty, very degenerate. Vascularity: slight; chrome: ditto. Perpendicular fibres: scanty, degenerate. Strong fibres less than in "A." A worse specimen than "A."

Summary.—Cross-fibres show considerable degeneration in parietal; these and radii are worse in one part examined than the other. Superradiary fibres are worse than tangential. The Baillarger band and strong and perpendicular fibres are better in the better specimens. Vascularity and chrome not increased at all commensurately with fibre-degeneration.

Occipital gyri, "A."—All cross-layers and radii: moderately degenerate and scanty. Superradiary: as bad as tangential. Terminals of radii: worse than trunks. Vascularity and chrome but slight.

Occipital gyri, "B" (another part).—Tangential fibres: fairly numerous, moderately degenerate. Superradiary: very scanty and very degenerate. Interradiary: fairly numerous, moderately degenerate. Radii: moderately scanty and moderately degenerate. Terminals: worse. Vascularity +, on one side chiefly. Chrome +, chiefly interradiary.

Summary.—Degeneration is chiefly moderate of all cross-layers; superradiary on the whole is worst; interradiary equally affected with tangential. Radii share in the moderate degeneration, and their terminals partake in the worse degeneration of superradiary area. Vascularity and chrome but slightly increased.

Temporal gyri.—Tangential fibres: numerous, good band, but fibres very degenerate. All other cross-fibres and also radii exceedingly scanty and degenerate, and absent in

various parts. Strong fibres in tangential band, but none elsewhere. No perpendicular. Vascularity and chrome both somewhat +, but especially so in foci, together.

Summary.—An exceedingly degenerate specimen, with great destruction of fibres, excepting tangential. Vascularity and chrome +, with particular destruction around.

General summary.—In this case of chronic mania with terminal dementia the cross-fibres throughout show considerable degeneration; the radii also. The temporal region is a good deal the worst, followed by the parietal and the occipital. Baillarger, strong, and perpendicular fibres are all better in the better specimens. The superradiary fibres are about the worst, and interradiary are as bad as tangential, even worse than these in the temporal area. The persistence of the tangential band, though composed of very degenerate fibres, in the temporal region, whilst other fibres are largely destroyed, is striking. Vascularity and chrome are +, especially in the worst (temporal) region; elsewhere the increase is not commensurate with fibre-degeneration.

(5) A. J.—On admission: subacute mania. Causation: stated to be shock, in connection with death of mother. Clinical course: subacute mania passing into dementia with periodic outbreaks of excitement and violence. At death: dementia. Cause of death: heart disease. Total duration: 40 years. Age at death: 59.

Frontal gyri.—Tangential fibres: very scanty, slight degeneration. Superradiary: ditto. Interradiary: ditto. Radii: only scanty. Vascularity: considerable; also chrome.

Summary.—All cross-layers but slightly degenerate. Radii probably not degenerate. Vascularity and chrome increased out of proportion to fibre-changes.

Parietal gyri.—Tangential fibres: scanty, slightly degenerate. Superradiary: normal. Interradiary: ditto. Radii: ditto. Vascularity and chrome = 0.

Summary.—Normal, except slight tangential changes.

Occipital gyri.—Tangential fibres: very scanty and moderately degenerate. Superradiary: ditto. Interradiary: scanty and very degenerate, the Gennari band thin over the

summit. Radii: very degenerate, including terminals (even worse). Vascularity +, capillary, and chiefly interradiary. Chrome: normal. Some perpendicular fibres, but very scanty and very degenerate.

Summary.—All cross-layers degenerate, but deepest layer most so, and radii even worse. Vascularity = + in worst fibre area. Chrome = 0.

Temporal gyri.—Tangential fibres: moderately degenerate. Superradiary: extremely scanty, very degenerate. Interradiary: ditto. Radii: only scanty. Vascularity and chrome + and marked.

Summary.—All cross-layers degenerate, the outermost least so. Radii escape.

General summary.—In this case of subacute mania with terminal dementia the cross-fibres show degeneration in all regions, though the parietal is almost normal; the temporal region perhaps the worst, though occipital is scarcely better; frontal but slightly affected. The interradiary layer is the worst in the occipital, the superradiary and interradiary in the temporal, the tangential being less affected. The radii are only scanty in the otherwise very degenerate temporal, very degenerate in the occipital. The normal area is neither vascular nor chromy, whilst the others show vascularity and chrome +.

(6) R. D.—On admission: acute mania. Causation: unknown. Clinical course: the condition became gradually chronic: dementia came on eight years after admission; remained a noisy chronic maniac, with weakness of mind, till death from pelvic abscess. Total duration: 33 years. Age at death: 54.

Frontal gyri.—Tangential fibres: numerous, moderately degenerate. Superradiary: numerous, very degenerate. Interradiary: numerous, moderately degenerate. Radii: normal. Vascularity and chrome = 0.

Summary.—All cross-layers show degeneration, especially superradiary. No scantiness of fibres. Otherwise normal.

Parietal gyri.—Tangential fibres: numerous, moderately degenerate. Superradiary: extremely scanty, moderately de-

generate. Interradiary: fairly numerous, slightly degenerate. Radii: normal; terminals degenerate. Vascularity and chrome = 0.

Summary.—All cross-layers show degeneration, especially superradiary; terminals of radii involved with them.

Occipital gyri.—Tangential fibres: extremely scanty (almost absent), very degenerate. Superradiary: fairly numerous, moderately degenerate; some strong fibres. Interradiary: not scanty, but very degenerate. Radii: not scanty, moderately degenerate. Vascularity and chrome +, latter chiefly about vessels.

Summary.—All cross-fibres degenerate, the outermost most so. Radii moderately involved. Vascularity and chrome + with fibre-degeneration.

Temporal gyri.—Tangential fibres: normal. Superradiary: very scanty (hardly exist), very degenerate. Interradiary: extremely scanty, moderately degenerate. Radii: fibres scanty only. Vascularity + and chrome much +.

Summary.—Tangential fibres: normal. Superradiary: much the worst of remaining layers. Vascularity and chrome + in conjunction with fibre-degeneration.

General summary.—In this case of chronic mania with terminal dementia the cross-fibres are considerably diseased throughout, principally the outermost two layers, in varying measure, with the exception of the tangential fibres in the temporal area, which are normal. The occipital region is worst; then follow in order: parietal, temporal, frontal. In the three last regions the superradiary layer is the worst. The radii are only moderately involved even in the worst specimens. Vascularity and chrome not always associated with fibre-degeneration, but they are increased in the worst specimens.

(7) L. P.—On admission: mania, with delusions of persecution. Causation: unknown (heredity present). Clinical course: delusions marked throughout; very hostile, raving frequently. At death: delusional insanity with dementia. Cause of death: phthisis. Total duration: $7\frac{3}{4}$ years. Age at death: 62.

Frontal gyri.—Tangential fibres: very scanty and very degenerate. Superradiary: scanty and very degenerate. Interradiary: scanty, moderate degeneration. Radii: very scanty, slightly degenerate. Vascularity + and chrome in patches.

Summary.—Degeneration of all cross-layers, especially of two outermost. Radii: involved in lesser degree, and vascularity and chrome +.

Parietal gyri.—Tangential fibres: scanty, very degenerate. Superradiary: very scanty, very degenerate. Interradiary: normal. Radii: scanty, slightly degenerate. Vascularity and chrome = 0.

Summary.—Degeneration of two outermost layers, and radii involved in minor degree. No commensurate increase of vascularity and chrome.

Occipital gyri.—Tangential fibres: very scanty, moderately degenerate. Superradiary: very scanty, moderately degenerate. Interradiary: scanty, slightly degenerate. Radii: scanty, moderately degenerate. Vascularity and chrome rather +.

Summary.—Cross-fibres: moderately degenerate, especially outer two layers. Radii: almost equally involved. Vascularity and chrome increased about proportionately.

Temporal gyri.—Tangential fibres: very scanty, moderately degenerate. Superradiary: very scanty (almost absent), very degenerate. Interradiary: scanty, moderately degenerate. Radii: scanty, very degenerate. Vascularity: much +, and chrome +.

Summary.—Cross-layers and radii all degenerate, the superradiary the worst. Vascularity and chrome proportionately increased.

General summary.—In this case of delusional insanity, with terminal dementia, the cross-fibres are degenerate, in considerable degree, in the different cerebral regions, the outermost two layers being chiefly affected. The superradiary is as much involved as the tangential. The radii are involved in a lesser degree, except in the temporal region (the worst), where they are equally affected with the worst of the cross-fibres. Vascularity and chrome are

associated with the fibre-degeneration. The temporal region is the worst, followed by the frontal, occipital, and parietal in order.

(8) J. M.—On admission: acute mania. Causation: heredity, otherwise unknown. Clinical course: acute mania with occasional stupor. At death: mania. Cause of death: exhaustion from mania. Total duration: $1\frac{1}{3}$ years. Age at death: 32.

Frontal gyri.—Tangential fibres: very scanty and very degenerate. Superradiary: ditto. Interradiary: ditto. Radii: numerous, moderately degenerated. Vascularity and chrome = 0.

Summary.—All cross-layers: very degenerate. Radii: moderately involved. No proportionate increase in vascularity and chrome.

Parietal gyri.—Tangential fibres: fairly numerous, very degenerate. Superradiary: ditto. Interradiary: normal. Radii: ditto. Vascularity and chrome = 0.

Summary.—The two outermost layers degenerate, otherwise normal.

Occipital gyri.—Tangential fibres: scanty, moderately degenerate. Superradiary: ditto. Interradiary: ditto. Radii: scanty, very slightly degenerate. Terminals: degenerate. Vascularity and chrome = 0.

Summary.—All cross-layers degenerate in moderate degree. Radii: only slightly affected, but their terminals more so. Vascularity and chrome not involved with fibre-changes.

Temporal gyri.—Tangential fibres: not scanty, moderately degenerate. Superradiary: almost absent, very degenerate. Interradiary: scanty and very degenerate. Radii: ditto. Vascularity and chrome = 0.

Summary.—All cross-layers degenerate, most so in deeper parts. Superradiary: worst. Radii: equally involved. Vascularity and chrome do not follow fibre-degeneration.

General summary.—In this case of acute mania the cross-fibres are not normal in any region, and are mostly notably degenerate. The frontal region is about the worst.

closely followed by the temporal; then come the parietal and occipital, about equally involved. The superradiary layer is as much affected as the tangential, and sometimes more so. The radii are less affected than the cross-fibres on the whole, though equally involved in the temporal region. There is a striking absence of vascularity and chrome.

(9) E. W.—On admission: delusional insanity (ideas of persecution). Causation unknown. Clinical course: remained same throughout, very hostile. At death: the same. Cause of death: aortic valvular disease. Total duration insanity: 6 years. Age at death: 37.

Frontal gyri.—Tangential fibres: extremely scanty and very degenerate. Superradiary: ditto. Interradiary: ditto. Radii: scanty and very degenerate. Vascularity +. Chrome = 0.

Summary.—All cross-layers and radii very degenerate. Vascularity is increased therewith.

Parietal gyri.—Tangential fibres: extremely scanty and very degenerate. Superradiary: ditto. Interradiary: ditto. Radii: scanty, very degenerate. Vascularity and chrome = 0.

Summary.—All cross-layers and radii very degenerate. No corresponding increase of vascularity and chrome.

Occipital gyri.—Tangential fibres: extremely scanty and very degenerate. Superradiary: ditto. Interradiary: ditto. Radii: ditto. Vascularity and chrome = 0.

Summary.—As in last.

General Summary.—In this case of delusional insanity the cross-fibres are extremely degenerate and the radii equally so. All parts equally bad. Vascularity and chrome are not increased in two of the regions, and their increase may be said to be not at all commensurate with fibre-degeneration.

(10) E. G.—On admission: acute melancholia. Discharged and remained out five to six years; readmitted in same state with some mental weakness also. Causation:

unknown. Clinical course: melancholia, passing into dementia. At death: dementia. Cause of death: chronic renal disease. Total duration: 14 to 15 years. Age at death: 72.

Parietal gyri.—Tangential fibres: very scanty, moderately degenerate. Superradiary: very scanty, moderately degenerate. Interradiary: normal. Radii: ditto. Vascularity and chrome = 0.

Summary.—The outermost two layers show moderate degeneration; otherwise normal.

Occipital gyri—Tangential fibres: very scanty and very degenerate. Superradiary: ditto. Interradiary: ditto. Radii: ditto. Vascularity and chrome = 0.

Summary.—All cross-layers and radii very degenerate, and equally so. Vascularity and chrome, on the contrary, not affected.

Temporal gyri.—Tangential fibres: not scanty, slightly degenerate. Superradiary: scanty, moderately degenerate, worse than former layer. Interradiary: ditto. Baillarger band exists. Radii: scanty and degenerate. Vascularity = 0. Chrome = rather + in superradiary and interradiary layers.

Summary.—The cross-fibres degenerate in varying degree, the tangential rather less than deeper ones. Radii involved equally with latter. No disease in vascularity but there is in chrome.

General Summary.—In this case of chronic melancholia, passing into dementia, the cross-fibres show varying degrees of degeneration (superradiary the worst), being worst in the occipital region, followed successively by the temporal and parietal regions. The radii pretty well follow suit, worst in occipital and normal in parietal. Vascularity and chrome do not follow fibre-degeneration, there being no vascularity at all and only very localised chrome.

(11) J. T.—On admission: subacute mania. Causation: no certain cause. Clinical course: subacute mania, passing into dementia shortly before death. Cause of death: broncho-pneumonia. Total duration: 6 months. Age at death: 58.

Frontal gyri.—Tangential fibres: rather scanty and very degenerate. Superradiary: fairly numerous, very degenerate. Interradiary: numerous, moderately degenerate. Radii: scanty, moderately degenerate; terminals more so. Vascularity and chrome = 0.

Summary.—Cross-fibres degenerate, especially the two outermost layers. Radii less degenerate than these but terminals equally bad. Vascularity and chrome not increased with fibre-changes.

Parietal gyri.—Tangential fibres: numerous but extremely degenerate. Superradiary: numerous, but less degenerate. Interradiary: numerous but less degenerate still. Radii: numerous; moderately degenerate. Vascularity = 0. Chrome only in interradiary.

Summary.—All cross-layers and radii show degeneration, worst and marked in outermost layer and progressively less from without. Vascularity and chrome not increased with fibre-degeneration.

Temporal gyri.—Tangential fibres: very scanty and very degenerate. Superradiary: ditto. Interradiary: numerous, moderately degenerate. Radii: scanty, moderately degenerate. Terminals very degenerate. Vascularity and chrome = 0.

Summary.—Cross-fibres degenerate, especially of outermost two layers. Radii less affected than these, but terminals equally bad. Vascularity and chrome not increased with fibre-changes.

General summary.—In this case of subacute mania, passing into dementia, the cross-fibres show considerable degeneration in all regions, the temporal being rather the worst, the other two regions about equally affected. In all parts the two outermost layers are most involved, the tangential being the worst in one. The radii are less affected but their terminals share in the degeneration of the layer in which they lie. Vascularity and chrome are singularly absent, notwithstanding fibre-degeneration.

(12) M. J.—On admission: subacute mania. Causation: unknown. Clinical course: recurrent mania; ultimately

some dementia. At death: in same mental state. Cause of death: general tuberculosis. Total duration: 31 years. Age at death: 52.

Frontal gyri.—Tangential fibres: scanty and very degenerate. Superradiary: extremely scanty and very degenerate (worse). Interradiary: ditto. Radii: very scanty and very degenerate. Vascularity = +, slightly.

Summary.—All cross-layers and radii very degenerate. Superradiary and probably interradiary worse than tangential. Vascularity is not commensurate with fibre-changes.

Parietal gyri.—Tangential fibres: fairly numerous and moderately degenerate. Superradiary: extremely scanty and very degenerate. Interradiary: numerous, but moderately degenerate. Radii: very degenerate, involved in chrome. Vascularity = 0, but chrome much +, especially interradiary.

Summary.—All cross-layers degenerate, superradiary the worst. Radii equally involved with the worst. Vascularity does not go with fibre-degeneration, but chrome is much +.

Occipital gyri.—Tangential fibres: fairly numerous, moderately degenerate. Superradiary: scanty and very degenerate. Interradiary: scanty, moderately degenerate. Radii: scanty, very degenerate. Vascularity = 0; chrome = +.

Summary.—All cross-layers show degeneration, superradiary the worst, next the interradiary, and radii equally degenerate with worst. Vascularity does not go with fibre-changes, but chrome does.

Temporal gyri.—Tangential fibres: fairly numerous, very degenerate. Superradiary: very scanty and very degenerate (worse). Interradiary: numerous, moderately degenerate. Radii: scanty, very degenerate. Vascularity = 0; chrome +, chiefly interradiary.

Summary.—All cross-layers degenerate, superradiary worst. Radii equally degenerate. Vascularity does not go with fibre-changes, but chrome does.

General summary.—In this case of recurrent mania with terminal dementia the cross-fibres and radii show considerable degeneration in the different parts of the hemisphere, the frontal area being the worst. It is noteworthy that in

all regions the superradiary layer is worse than the tangential or interradiary, and the latter is nearly as much affected as the tangential. Further, the radii are equally degenerate with the cross-fibres. With much fibre-degeneration there is little or no vascular increase, whilst chrome is increased.

(13) H. D.—On admission: chronic melancholia with some dementia. Causation: not clear; came on gradually. Clinical course: the same condition until death, which resulted from cardiac mural disease. Total duration: $3\frac{1}{2}$ years. Age at death: 57.

Frontal gyri.—Tangential fibres: not scanty, moderately degenerate. Superradiary: scanty, very degenerate. Interradiary: ditto. Radii: scanty, moderately degenerate; terminals very much so. Vascularity = 0; chrome + slightly.

Summary.—All cross-layers show degeneration; superradiary and interradiary most, and equally; radii trunks moderately, terminals greatly, affected. Vascularity not increased with fibre changes.

Parietal gyri.—Tangential fibres: not scanty, moderately degenerate. Superradiary: scanty, very degenerate. Interradiary: fairly numerous, moderately degenerate. Radii: scanty, very degenerate. Vascularity = 0; chrome +, slightly.

Summary.—All cross-layers show degeneration, superradiary most, interradiary as much as tangential. Radii as affected as superradiary. Vascularity not increased with fibre changes.

Temporal gyri.—Tangential fibres: not scanty, very degenerate. Superradiary: scanty, moderately degenerate. Interradiary: numerous, moderately degenerate. Radii: very scanty and very degenerate. Vascularity = 0; chrome +, slightly.

Summary.—All cross-layers show degeneration, tangential the most. Radii are even worse than cross-layers. Vascularity not increased with fibre-changes.

General summary.—In this case of chronic melancholia with some dementia the cross-fibres and radii show considerable degeneration in the different regions examined, which

are about equally affected. The superradiary affected to a rather greater degree than the other cross-fibres, interradiary as much diseased as tangential. The radii are diseased in an extraordinary degree, equally with the worst cross-fibres. With much fibre-degeneration no increase in vascularity, and chrome also shows slight increase.

(14) L. B.—On admission: acute mania. Causation: the puerperal state. Clinical course: acute mania throughout. At death: the same. Cause of death: exhaustion and acute enteritis. Total duration: $5\frac{1}{2}$ months. Age at death: 26.

Frontal gyri.—Tangential fibres: numerous, moderately degenerate. Superradiary: ditto. Interradiary: normal. Radii: ditto. Vascularity = 0; chrome +, general.

Summary.—Normal, with exception of moderate degeneration of two outermost layers.

Parietal gyri.—Tangential fibres: normal. Superradiary: slightly degenerate. Interradiary: normal. Radii: ditto. Normal perpendiculars. Vascularity and chrome = 0.

Summary.—Normal, except from slight superradiary degeneration.

Occipital gyri.—Tangential fibres: numerous, very degenerate. Superradiary: ditto. Interradiary: ditto. Radii: extremely degenerate and scanty. Vascularity = 0; chrome +, general, deep.

Summary.—All cross-layers show much degeneration, though fibres are numerous. Radii are rather worse. No increase in vascularity, but chrome is +.

Temporal gyri.—Tangential fibres: normal. Superradiary: ditto. Interradiary: ditto. Radii: scanty, moderately degenerate; terminals normal. Vascularity and chrome = 0.

Summary.—Cross-layers normal. Radii moderately degenerate. Vascularity and chrome not present.

General summary.—In this case of acute puerperal mania the occipital region shows the most degeneration by far, the frontal comes next. The parietal and temporal are not far from normal, the latter showing degenerate radii and normal cross-layers—a very unusual condition. On the whole the

outermost two layers are most affected, superradiary as much as tangential. Vascularity is not present in conjunction with fibre-degeneration, whereas chrome is, in the most degene rate parts.

(15) L. B.—On admission: acute melancholia. Causation: the puerperal state. Clinical course: acute melancholia throughout. At death: the same. Cause of death: exhaustion. Age at death: 23. Total duration, $2\frac{1}{2}$ years.

Frontal gyri.—Tangential fibres: extremely scanty and very degenerate. Superradiary: ditto. Interradiary: ditto. Radii: scanty and very degenerate. Vascularity and chrome +.

Summary.—All cross-layers and radii very degenerate, with vascularity and chrome increased proportionately.

Parietal gyri.—Tangential fibres: scanty and degenerate. Superradiary: ditto. Interradiary: normal. Radii: ditto. Vascularity +, slight; chrome = 0.

Summary.—The two outermost cross-layers degenerate, but less than last. Vascularity in accordance with fibre-changes.

Temporal gyri.—Tangential fibres: very scanty and moderately degenerate. Superradiary: scanty and degenerate. Interradiary: ditto. Radii: scanty and very degenerate. Vascularity +, slight; chrome = 0.

Summary.—All cross layers show degeneration, chiefly outermost, but more marked. Radii: rather worse. Vascularity only slight.

Occipital gyri.—Tangential fibres: very scanty and very degenerate. Superradiary: numerous, very degenerate. Interradiary: numerous, degenerate. Radii: numerous, slightly degenerate. Terminals extremely so. Vascularity and chrome only locally + (superradiary).

Summary.—All cross-layers show degeneration, especially outermost. Terminals of radii involved with superradiary. Vascularity and chrome scarcely proportionately increased.

General summary. — In this case of acute puerperal melancholia the cross-fibres show considerable degeneration in the various parts of the hemisphere examined (especially

the outermost two layers), the frontal region being the worst, the parietal showing least, the other two regions about equally affected. The radii show considerable degeneration in the worst area, but escape in the part with least degeneration. Vascularity and chrome are increased in the worst area, but do not go *pari passu* with fibre changes in the other parts.

(16) A. J.—On admission: paralytic dementia, demented type. Causation: heredity +, ? alcoholism. Clinical course: demented type of general paralysis throughout, resistive, helpless. At death: ditto. Cause of death: paralytic dementia. Age at death: 53. Total duration: about 10 months.

Frontal gyri (6).—Tangential fibres: scanty and very degenerate, with some variation in degree in different gyri. Superradiary: very scanty and very degenerate in all; worse than last. Interradiary: ditto. Radii: good in some gyri, but very degenerate on the whole. Vascularity = + in majority gyri; two are not vascular. Chrome = + in all.

Summary.—All cross-layers show much degeneration with some variation in degree in different gyri. Superradiary and interradiary the worst, and equally. Radii: usually very degenerate. Vascularity and chrome: generally + with fibre-degeneration.

Sulci (5).—Connected with the gyri. The cross-fibres are in general numerous, and show moderate degeneration, especially outermost layer. Variations between different sulci. Vascularity and chrome + in all save one.

Parietal gyri (5).—Tangential fibres: variations exist in different gyri and also in different parts of individual gyri. Generally degenerate; most over summit. Superradiary: ditto. Interradiary: not scanty but degenerate. Radii: generally degenerate; the same variations. Baillarger well-marked in most gyri. Terminals worse. Vascularity +, capillary uniform. Chrome only slightly +. Perpendicular fibres exist; very degenerate. One gyrus normal.

Summary.—General degeneration of cross-layers and radii, with variations in degree in different gyri and in

different parts of individual gyri. Summit of gyrus generally the worst site. Vascularity +, but chrome less so. Persistence of Baillarger. A normal gyrus present with degenerate.

Sulci (4).—The fibres of the two outermost layers scanty and degenerate, those of interradiary vary, some being normal. Vascularity +, capillary.

Occipital gyri (4).—Tangential fibres: in general very scanty and very degenerate. Variations similar to last. Superradiary: in all very scanty and degenerate. Interradiary: in general very scanty and degenerate. Radii: scanty and degenerate in two, normal in two. Terminals only escape in one. Vascularity +, chiefly superradiary. Chrome + in some, not in all.

Summary.—General degeneration of cross-layers, with variations between gyri. Radii less generally affected. Vascularity + with fibre-changes. Chrome is more variable.

Sulci (5).—Fibres hardly exist, and are very degenerate in majority. Normal in remainder. Vascularity = + in some, none in others, without reference to fibre degeneration.

General summary.—In this case of paralytic dementia the cross-layers and the radii are much degenerated in the different parts of the hemisphere. The cross-layers about equally involved. Radii scarcely better. Variations between neighbouring gyri and in individual gyri in different parts. The different parts about equally affected, though frontal more uniformly than others. Vascularity = + with fibre-changes. Chrome is also +, but more variable in this respect. As regards sulci there is much variation; the majority show degeneration, but not so much as in gyri. Some are normal. Most show vascularity and chrome +.

(17) J. H.—On admission: paralytic dementia, maniacal type. Causation: not clear; had been seafaring man, and alcohol very probable. Clinical course: maniacal type; general paralysis throughout. At death: demented; died in convulsions. Cause of death: general paralysis of the insane. Age at death: 54. Total duration: 2 years.

Frontal "A."—Tangential fibres: very scanty, very degenerate. Superradiary: ditto. Interradiary: ditto. Radii: ditto. Vascularity and chrome + (extreme).

Summary.—All fibres, cross and radii, very degenerate. Vascularity and chrome associated therewith.

Frontal "B" (another part).—Tangential fibres: extremely scanty and extremely degenerate. Superradiary: ditto. Interradiary: ditto. Radii: very scanty and very degenerate. Vascularity and chrome + (extreme).

Summary.—Rather worse than "A."

Frontal "C" (another part).—Tangential fibres: extremely scanty and extremely degenerate. Superradiary: ditto. Interradiary: ditto. Radii: ditto. Vascularity and chrome + (extreme).

Summary.—As in "B."

Frontal "D" (another part) *gyri* (5).—Tangential fibres: absent. Superradiary: ditto. Interradiary: ditto. Radii: scanty and degenerate. Vascularity and chrome + (extreme). Chrome around the vessels especially.

Summary.—A worse specimen than others. Cross-fibres: absent. Radii: less affected. Vascularity and chrome go with fibre-changes.

Summary for frontal area.—Very marked degeneration of cross-layers and radii, with some variations in different parts of the lobe. The cross-fibres absent in some parts. Vascularity and chrome greatly +, with fibre-degeneration.

Sulci (5).—Except very scanty and very degenerate intermediary, no cross-fibres. Vascularity and chrome + in all.

Parietal gyri (4).—Tangential fibres: very scanty and very degenerate. Variations in different gyri and in individual gyri. Superradiary: ditto. Interradiary: very scanty, slight degeneration; variations as above. Baillarger: well marked. Radii: normal in two, scanty and degenerate in other two. Vascularity and chrome greatly + (latter around vessels).

Summary.—All cross-layers show degeneration, marked in outer two. Variations between gyri and parts of a gyrus. Radii: less affected. Vascularity and chrome + with fibre-degeneration.

Sulci (3 and 4).—Fibres degenerate. Vascularity and chrome +.

Occipital gyri (4).—Tangential fibres almost absent, very degenerate. Superradiary: ditto. Interradiarry: much the same, but variations between gyri. Radii: extremely scanty and very degenerate. Variations as above. Vascularity and chrome +, latter around vessels.

Summary.—All cross-layers show great degeneration, interradiary less consistently than others; latter remark also applies to radii. Vascularity and chrome + with fibre changes.

Sulci (5).—Fibres extremely degenerate and vascularity and chrome +, slightly.

Temporal gyri (5).—Tangential fibres: absent. Superradiary: ditto. Interradiary: scanty and extremely degenerate. Radii: ditto. Vascularity and chrome greatly +, latter about vessels.

Summary.—Extreme degeneration of all medullated fibres. Vascularity and chrome + in proportion.

Sulci (3 and 4).—Vascularity and chrome greatly +. Fibres absent, except very few and degenerate interradiary.

General summary.—In this case of paralytic dementia the medullated fibres, especially the cross-fibres, show very marked degeneration (to the extent of complete destruction sometimes) in all regions of the hemisphere, without appreciable distinction, except for the parietal, which is rather less affected. At the site of greatest disease, in a given area, the gravity of the disease does not allow of any variations between gyri and parts of these; elsewhere these variations are seen. The degeneration is so great that Baillarger's band is no longer to be made out, except rarely, where the conditions are not so extreme (parietal). On the whole, the two outermost layers are the worst parts of the cortex, and are equally involved. Vascular disease, extreme and general, with fibre-degeneration. Chrome greatly + around vessels.

In the *sulci* the fibre-degeneration and vascularity and chrome are about equal to the gyri.

(18) G. E. On admission: imbecile (noisy, violent, impulsive). Causation: heredity, intensified by alcoholic

habits. Clinical course: the same throughout. At death: the same. Cause of death: Landry's paralysis (ten days' duration). Age at death: 30.

Frontal gyri (3).—Tangential fibres: scanty, extremely degenerate. No notable variations between different gyri; local variations in individual cases. Superradiary: very scanty, extremely degenerate. Some variations. Interradiary: ditto. Baillarger present. Radii: very scanty and extremely degenerate; worst over summit. Vascularity +, also chrome, but not always together, and not general.

Summary.—All cross-layers and radii show extreme degeneration with local variations in degree within the gyrus. Baillarger band escapes the other interradiary degeneration. Vascularity and chrome +, but not general.

Sulci.—Fibres degenerate and scanty. Interradiary: better than others. Vascularity and chrome +.

Central "A" gyri (3).—Tangential fibres: slightly scanty. moderately degenerate; worse at summit. Superradiary: very scanty and very degenerate. Interradiary: slightly scanty, very degenerate. Variations between gyri. Radii: scanty and degenerate; worse over summit. Vascularity and chrome: slight.

Summary.—All cross-layers and radii show degeneration, the superradiary about the worst. Conditions rather worse at summit. The interradiary vary in degeneration in different gyri. Vascularity and chrome: slight, and not in proportion to above changes.

Central "B" (another part) *gyri* (3).—Tangential fibres: scanty and moderately degenerate. Variations noted in individual gyri. Superradiary: scanty and very degenerate. Interradiary: scanty and degenerate. Radii: scanty and degenerate; terminals worse. Vascularity +, chiefly interradiary, and chrome + about vessels.

Summary.—All cross-layers and radii scanty and degenerate, with superradiary about the worst. Variations as respects tangential: as above. Vascularity and chrome +, localised.

Summary, central.—All cross-layers and radii show degeneration, superradiary rather the worst. Some varia-

tions between gyri and in gyri, but minor. Vascularity and chrome not striking; scarcely proportionate to other changes.

Sulci (3).—Fibre-degeneration, two outermost layers especially. Vascularity +. Chrome = 0.

Occipital "A" gyri (7).—Tangential fibres: very scanty and very degenerate. Gyri equally bad. Variations between gyri and between parts of individual gyri, generally worst over summit. Superradiary: very scanty and very degenerate. Gyri: unequally affected. Interradiary: scanty, moderately affected. Variations as above. One gyrus normal. Radii: scanty and degenerate, especially terminals. Two gyri normal. Vascularity + greatly, and worst with fibre-changes. Chrome is more irregular. Strong fibres absent.

Summary.—The cross-layers are very degenerate, especially the outer two. Variations in degree as above. Radii: mostly degenerate, and terminals worse. Vascularity + with above changes.

Occipital "B."—A similar summary to "A." Vascularity is +, but with variations in different gyri.

Sulci (7).—Fibres normal. Vascularity +; chrome much less so.

Temporal gyri (5).—Tangential fibres: much variation, some normal, some very scanty and degenerate in parts of a gyrus. Superradiary: ditto. Interradiary: normal. Radii: ditto. Vascularity = +, chiefly outermost layers. Chrome +, in deeper layers.

Summary.—A better specimen than the preceding; some gyri normal; in others the two outermost layers show degeneration with variations between gyri and in individual gyri. Vascularity + in affected parts; chrome +, but does not follow vascularity.

Sulcus (1).—Fibres normal; vascularity only slight.

General summary.—In this case of imbecility with excess in alcohol the medullated fibres show considerable degeneration, the frontal and central regions worse than the occipital and temporal: the first-named most, the last least. There are variations between gyri and parts of individual gyri in the different regions. Vascularity is generally + in con-

junction with fibre-degeneration, but not a striking feature. Chrome is not marked and not regularly associated with the vascular change. As regards *sulci*, the fibres are the worst in those connected with the worst gyri—frontal and central: and vascularity and chrome most marked in the same.

(19) J. W. On admission: paralytic dementia, maniacal type. Causation: overstrain. Clinical course: maniacal type, general paralysis until death. Total duration: 5½ months. Age at death: 37.

Frontal "A" *gyri* (5).—Tangential fibres: extremely scant, extremely degenerate all through. Superradiary: ditto, but rather less scanty. Interradiary: ditto, one gyrus rather less scanty. Radii: scanty, moderately degenerate, terminals very degenerate, one gyrus worst. Vascularity greatly +, chiefly super- and interradiary. Chrome: ditto, obscures all in places. Pia greatly thickened and infiltrated with round cells. Strong fibres practically absent.

Summary. — Extreme degeneration of cross-layers throughout, tangential rather the worst. Radii less affected, excepting terminals much so. Vascularity and chrome go with fibre-changes.

Frontal "A^1" (same part, 5 gyri). All as above. One gyrus shows normal radii.

Frontal "B" (another part, 1 gyrus).—Fibres and radii practically as in last two. Superradiary worse than tangential. Vascularity +, chiefly outermost two layers. Chrome: slight, diffuse.

Summary.—Same degenerations as "A"; superradiary worst. Vascularity has rather different seat of election: chrome is less than in "A."

Frontal "C" (another part, 2 gyri).—Tangential fibres as in previous in one gyrus, better in the other. Superradiary: as in previous. Interradiary: ditto. Radii: normal. Vascularity and chrome +.

Summary.—All cross-layers degenerate; tangential shows variations. Radii: normal. Vascularity and chrome go with fibre-changes.

Frontal "D" (another part, 2 gyri).—Cross-layers and

radii: as in "A," and even worse conditions obtain. Two outermost the worst.

Summary, frontal region.—Degeneration of cross-fibres is great throughout, variations unimportant. The two outermost layers are the worst, but superradiary are as affected, at least, as tangential. Radii not so regularly or extensively affected as the cross-layers. Vascularity and chrome commonly much +, with varying seats of greatest intensity. Strong and perpendicular fibres are practically absent.

In seven *sulci* examined the fibres are very scanty and degenerate, the absence of the sweep of external association fibres of Meynert in some sulci is striking. Vascularity and chrome +.

Frontal near ascending frontal "A" (5 gyri).—Tangential fibres: very scanty and very degenerate in all, with variations in degree, in difference, and in individual gyri. Superradial: very scanty and very degenerate. Interradiary: fairly numerous, moderately degenerate. Radii: majority normal as regards trunks, but terminals are degenerate; minority very scanty and degenerate. Vascularity and chrome + in all.

Summary.—Cross-layers degenerate in all, the two outermost chiefly, with variations, as above. Radii less affected, but variations, and terminals always involved. Vascularity and chrome go with the fibre-changes.

Sulci (2).—Tangential fibres: scanty and degenerate. Vascularity and chrome +.

Frontal near ascending frontal "B" (another part, 5 gyri).—Tangential fibres: very scanty and very degenerate in all, with variations between gyri. Superradiary: ditto. Interradiary: not scanty but degenerate, but worse in these respects in a minority. Extreme tortuosity in one gyrus. Baillarger well marked in some. Radii: numerous in three, scanty in two; varying degrees of degeneration. Normal in one gyrus. Vascularity greatly +, with variations in different parts individual gyri. Chrome: ditto.

Summary.—All cross-fibres show great degeneration, especially outermost two, with variations as described. Radii show less. Vascularity and chrome follow fibre-changes.

Sulci (3 and 4).—Fibres degenerate, involving interradiary in worst cases. Vascularity + in all, but chrome only in some.

Summary of frontal near ascending frontal.—Great degeneration of all cross-layers, especially of outermost two layers, with variations in degree between different gyri and locally in individual gyri. Radii: generally but not universally affected, and less than previous. Terminals worst. Strong and perpendicular fibres are scanty and degenerate. Vascularity and chrome +, with fibre-degeneration.

Sulci.—The fibres degenerate in general, but less notably.

Central "A" (5 gyri).—Tangential fibres: fairly numerous, all degenerate in different degrees in different gyri. Much worse in No. 5. Superradiary: scanty and degenerate, with similar variations, worst in No. 5; Nos. 2 and 4 normal. Interradiary: variations in gyri 1, 2, 4 normal, other two scanty and degenerate; No. 5 worst. Radii: ditto. Vascularity and chrome + in all in different degrees and varying intensity, even in same gyrus.

Summary.—The tangential layer is the worst, next superradiary. In some gyri they and interradiary are normal. Radii: also only degenerate in minority. Variations in different gyri for all fibres. The same gyrus worst in all. Vascularity and chrome +, and follow fibre-changes in the main.

Sulci.—Much as gyri; in some fibres degenerate, in others sound. Vascularity and chrome +.

Central "B" (another part, 5 gyri).—Tangential fibres: degenerate as a whole, not always scanty. No. 1 not degenerate. Variations in different gyri and parts of individual gyri. Superradiary: ditto. Interradiary: normal, and good Baillarger. Radii: three normal, two degenerate and scanty. Vascularity and chrome +, with variations, as for fibres.

Summary.—One gyrus normal throughout. The two outermost layers are affected equally, with variations as described. Radii: more affected than interradiary. Vascularity and chrome follow fibre-changes. Perpendicular fibres appear in the best gyri.

Sulci.—One normal; others show fibre-degeneration in two outermost layers. Vascularity and chrome + in varying degree.

Summary of central.—The two outermost cross-layers show degeneration, with variations in degree as described; the tangential sometimes the worst; in rest they are equally affected. The interradiary are principally normal, and more so in one part of the region than the other. The radii normal in majority of gyri, but quite as much affected as interradiary. Vascularity and chrome +, and follow fibre-degeneration. Most sulci show degeneration of fibres, especially of outermost layers, and vascularity and chrome +.

Parietal near occipital "A" (6 gyri).—Tangential fibres: very scanty and very degenerate in all; some gyri worse than others. Superradiary: ditto, and some gyri worse. Interradiary: ditto. Radii: Vascularity and chrome greatly +, and in association. All fibres worst near the chromo-vascular patches. No perpendicular fibres.

Summary.—All medullated fibres very degenerate and equally, the same gyri being worse in all respects. Vascularity and chrome +, with fibre-changes.

Sulci.—Fibres very degenerate, especially two outermost layers; superradiary worst. Vascularity +, also chrome, but less so.

Parietal near occipital "A^1" (same part, 7 gyri).—Bears out the preceding.

Parietal near occipital "B" (another part, 7 gyri).—Tangential fibres: scanty and degenerate in all. Superradiary: extremely scanty and degenerate in all; worse than tangential. Interradiary: very scanty and very degenerate in all. Baillarger persists more than others. Radii: very scanty and very degenerate throughout; some more than others. Vascularity greatly + throughout. Chrome +, but with variations. All fibres worse near vascular and chromy patches. No perpendicular.

Summary.—The cross-layers are very degenerate throughout, and so are the radii, though not so consistently, as there are variations. Superradiary: worse than tangential, and interradiary quite as bad as latter. The greater persistence

of Baillarger is to be noted. Vascularity and chrome are greatly +, but not always associated.

Sulci.—Fibres degenerate, especially outermost two layers, sometimes absent. Vascularity and chrome +. Inflammatory patches occur.

Summary of parietal near occipital.—Great degeneration of cross-layers and radii, with some variations in different gyri. The superradiary is sometimes the worst layer. Vascularity and chrome greatly +, and the nerve-fibres are worst in association with the areæ of vascularity and chrome. Perpendicular fibres partake in the general degeneration. Baillarger's layer shows greater persistence than rest of interradiary fibres. The sulci show similar conditions as to fibre, vascularity and chrome; the two outermost layers are the worst.

Occipital near tip "A" (9 gyri).—Tangential fibres: normal in four gyri, degenerate in others, with variations between gyri, and locally in individual gyri. Superradiary: same; normal in same and most in same, with variations as above. Interradiary: ditto. Radii: ditto. The worst fibres are in connection with engorged vessels and chrome patches. Perpendicular fibres: worst in worst gyri. Genuari's band is good in the best. Many strong fibres in normal parts. The gyri with best fibres are not vascular; others are unduly vascular and vary. Chrome in excess in all but two gyri; very variable distribution.

Summary.—The cross-layers less degenerate than in preceding areæ, being normal in four out of nine gyri. In the degenerate gyri variations occur, as above described. Same remarks apply to radii. Genuari's band strong and perpendicular fibres present in good specimens. Vascularity and chrome +, and follow fibre-degeneration, the latter less regularly.

Sulci (12).—Five normal as regards fibres and vascularity and chrome. Rest show degeneration, especially of outermost two layers; vascularity and chrome +, irregularly distributed.

Occipital near tip "B" (another part: 9 gyri).—Tangential fibres: three normal, others moderately degenerate;

summit of gyri worse, and variations between and in gyri. Superradiary: five normal, including above three; remainder moderately degenerate, with variations. Interradiary: normal throughout. Radii: normal in seven; others moderately degenerate. Vascularity +, goes with fibre-degeneration. Chrome +, with vascularity, but less regular distribution. Perpendicular fibres: thin and scanty.

Summary.—The cross-layers are less degenerate than in previous specimens, including occipital "A"; only outermost two layers affected, and tangential worse. Variations in degree, as above. The radii affected in limited degree. Vascularity and chrome +, chiefly with fibre-degeneration.

Sulci.—Normal, except two. In these only tangential fibres affected. Vascularity = + in both.

Summary of occipital.—The cross-layers and radii are less degenerate than in former parts of the brain examined, and less in some parts of the occipital than in others. Several normal gyri. Variations in degree of degeneration exist in the affected gyri and in different parts of individual gyri. Superradiary less affected than tangential, in some cases never more affected. Interradiary layer normal, and radii practically the same. Vascularity + with fibre-degeneration, and chrome also, but more irregular distribution.

Sulci.—Much as gyri; several normal, others show degeneration, especially of outermost two layers. Vascularity and chrome as in gyri.

Temporal "A" (7 gyri).—Tangential fibres: very scanty and extremely degenerate; two gyri show variations in degree within the gyrus. Superradiary: very scanty and very degenerate; variations between and in gyri. Interradiary: ditto. Radii: ditto. Vascularity and chrome + in all, chiefly in deeper parts.

Summary.—Cross-layers and radii everywhere very degenerate, with variations in different gyri, and within gyri. Vascularity and chrome +, and go with degeneration. Perpendicular fibres very scanty and degenerate.

Sulci (6).—Vascularity and chrome +. Fibres of outermost two layers degenerate in all, and interradiary ditto in three.

Temporal "B" (another part: 7 gyri).—Tangential fibres: extremely scanty and extremely degenerate. Variations between and in gyri, summit worst. Superradiary: ditto. Interradiary: ditto. Radii: scanty, very degenerate. Vascularity and chrome +. Perpendicular fibres thin and tortuous.

Summary.—A very bad specimen. Extreme degeneration of cross-layers, and radii is not much better. Variations in degree, as in last; but this specimen is worse than temporal "A."

Temporal "B" (same part: 7 gyri).—The same conditions as the last. Superradiary fibres are the worst. Sulci as in last.

Summary of temporal.—Extreme degeneration of cross-layers and radii, with variations in different and in individual gyri. Vascularity and chrome +, in association with fibre-degeneration. The conditions are worse than in occipital region, and worse in some parts of temporal than in others. The sulci rather less affected, the two outermost layers chiefly. Vascularity and chrome +.

General summary.—In this case of paralytic dementia of the "florid" type there is widespread and marked degeneration of all the cortical medullated fibres. The anterior portion of the frontal lobe and the mid-temporal region show the most marked degeneration, and one appears to be as bad as the other. Next comes the parietal near the occipital, thereafter the frontal near the descending frontal, then the central, and lastly the occipital region, with moderate degeneration and several normal gyri. Some parts of a region or lobe are worse than another, and variations in degree of degeneration between different, and even between different parts of individual, gyri are noticed. The two outermost layers are often the worst, sometimes equally bad, sometimes one, sometimes the other, worse. There is an absence of strong and of perpendicular fibres in the worst parts, whereas in the normal gyri they are present. Baillarger's band is also noted in normal gyri, with a greater persistence than other interradiary, in some gyri where the interradiary are involved in degeneration. The radii on the whole follow

the cross-fibres in degeneration, being worst where latter are worst, and *vice versâ*. But in the frontal region they are not so notably degenerated as in the temporal. In the least affected (central and occipital) they are mostly normal, and not extremely affected where diseased. Of vascularity and chrome it may be said that they are greatly increased, the vascularity being often enormous, and they vary directly with the degree of fibre-degeneration. Fibres are commonly worst in connection with foci of vascularity and chrome. The sulci show fibre-degeneration and increase of vascularity and chrome, usually much in same degree as the gyri with which they are associated. The absence (destruction) of Meynert's external association fibres (with the outermost layers) in the sulci of frontal specimens is striking, as this is very uncommon.

(20) A. W.—On admission: mania. Causation: heredity alone assigned. Clinical course: continued mania with much eroticism, passing into chronic mania which, with superadded mental weakness, was present at death. Cause of death: pneumonia. Total duration: 30 years. Age at death: 73.

Frontal (*pre-central*) "A" (6 gyri).—Tangential fibres: scanty, moderately degenerate throughout, with variations in different gyri and in individual ones. Superradiary: scanty and degenerate in all; similar variations. Interradiary: scanty and degenerate; two worse than others. Radii: normal in two, degenerate in varying degrees in others. Vascularity and chrome +, together with variations in different gyri. Perpendicular fibres degenerate; Baillarger fibres not distinguishable.

Summary.—Cross-fibres show moderate degeneration. Radii less generally affected. Variations in degree of degeneration. Vascularity and chrome +, with same variations.

Sulci (5).—Fibres moderately degenerate. Vascularity slight on the whole. Chrome increased in some.

Pre-central "A" (same part: 6 gyri).—Bears out previous specimens. No perpendicular and no Baillarger fibres observed.

Summary pre-central.—See under "A."

Parietal "A" (4 gyri).—Tangential fibres: rather scanty and moderately degenerate; some strong ones; variations in different and in individual gyri. Superradiary: ditto. Interradiary: two normal, with strong fibres; others scanty and moderately degenerate. Baillarger band good. Radii: moderately degenerate and rather scanty. Vascularity = 0 in three, + in one. Chrome + in three.

Summary.—The two outermost cross-layers worst, but only moderately degenerate. Variations as described. Interradiary less affected, but radii as much. Vascularity not increased in general, but chrome is.

Sulci (4).—Fibres mostly degenerate. Normal in one. Vascularity = 0; chrome +.

Parietal "B" (another part: 3 gyri).—Tangential fibres: scanty and degenerate, with variations as described in last. Superradiary: one normal, others very scanty and degenerate. Interradiary: two normal, the third degenerate. Baillarger layer good where fibres normal. Radii: same as interradiary. Vascularity = 0; chrome +, moderately and interradiary. Perpendicular fibres numerous and moderately degenerate.

Summary.—The cross-fibres and radii show degeneration, moderate; the two outermost layers more uniformly, and tangential most so. Vascularity = 0; chrome moderately and locally +.

Sulci.—Fibres scanty and degenerate. Vascularity and chrome = 0.

Summary parietal.—The cross-fibres and radii are moderately degenerate, especially two outermost layers, with variations in different and in individual gyri. A rather better specimen than the pre-central. Vascularity is not increased; chrome moderately so.

Sulci.—Fibres mostly scanty and degenerate. Vascularity and chrome not increased.

Occipital "A" (15 gyri).—Tangential fibres: normal in six; degenerate to moderate extent, and with variations between and in gyri in nine. Superradiary: normal in seven; moderately degenerate in eight, with same varia-

tions. Interradiary: normal in four; moderately degenerate and scanty, with variations, in eleven. Radii: normal in six, in nine scanty and moderately degenerate, with variations. Vascularity + in all save two. Chrome + moderately. Variations as in fibre-degeneration. Perpendicular fibres normal.

Summary.—The cross-layers and radii are normal in a large number of gyri and show only moderate degeneration in the others. No one layer worse than another. Variations in degree of degeneration, including local variations in individual gyrus, in all layers. Vascularity and chrome are +, especially former, with the same variations. Perpendicular fibres not affected.

Sulci.—Majority show scanty and degenerate fibres with vascularity and chrome +. Only one normal one.

Occipital "A^1" (same part, 11 gyri).—Tangential fibres normal in nine gyri, the other two show extreme degeneration in patches, in connection with vascularity and chrome. Superradiary: eight normal, the rest as in tangential fibres. Interradiary: same as superradiary. Radii: ditto. Vascularity and chrome normal in three, in eight they are +, with variations in different gyri.

Summary.—Greater part of specimen is normal; the two diseased gyri show extensive degeneration, all layers equally involved. Vascularity and chrome are +, out of proportion to the fibre-degeneration. Perpendicular fibres moderately degenerate.

Sulci (11).—Five normal. Others show fibre-degeneration and patchy vascularity and chrome.

Occipital "A^2" (same part, 7 gyri).—Tangential fibres normal in four, the others moderately degenerate, with variations between and in gyri. Superradiary: ditto. Interradiary: two normal, and others also except for foci of fibre-degeneration in connection with vascularity and chrome. Radii: five normal, others show local degeneration as above. Perpendicular fibres moderately degenerate, Genuari's band only prominent in one gyrus. Vascularity and chrome + in six, chiefly patchy, and variations in gyri, as above.

Summary.—More widespread, though moderate, degenera-

tion amongst cross-layers than in last. Some gyri normal. Radii principally normal. The degeneration is chiefly focal, in connection with vascularity and chrome. Variations in degree as described.

Sulci (4).—Fibres degenerate, interradiary least. Vascularity and chrome +.

Summary of occipital "A."—Only moderate degeneration of cross-layers and of radii in equal measure, and many gyri normal. Degeneration chiefly focal, in connection with vascularity and chrome +, in patches.

Sulci.—Fibres degenerate: fewer normal than *sulci.* Vascularity and chrome +.

Occipital "B" (another part: 5 gyri).—Tangential fibres normal, except for focal degeneration in connection with increase of vessels and connective tissue. Variations in gyri. Superradiary: normal. Interradiary: ditto in three, foci of degeneration in others. Radii: ditto. Vascularity and chrome +, in patches. Gyri four and five worst in all respects.

Summary.—Moderate degeneration of cross-layers and of radii in foci, with increase of vascularity and chrome: apart from these the specimens are normal.

Sulci (2).—Fibres degenerate and vascularity and chrome +.

Summary of Occipital.—The cross-layers and radii are normal in a large number of the gyri examined, but in about an equal number there is moderate degeneration, all layers being about equally involved. Variations in degree of degeneration between gyri and in gyri. The chief feature is the focal degeneration in connection with increase of vascularity and chrome in patches.

Sulci.—More show degeneration (of moderate degree) than in case of gyri. Vascularity and chrome +.

Temporal (7 gyri).—Tangential fibres extremely scanty and degenerate in all, with similar variations to last. Superradiary: three normal, four degenerate and scanty. Interradiary: all normal. Good Baillarger. Radii: ditto. Vascularity and chrome + slightly, with variations in different gyri.

Summary.—The two outermost cross-layers show degeneration, the outermost being the worst and most widespread. Varations in degree of degeneration as described. Vascularity and chrome slight, probably because the deeper parts are normal.

Sulci.—Fibres normal. Vascularity and chrome only slight.

General Summary.—In this case of chronic mania the cross-layers are only moderately degenerate in the different regions examined, the pre-central area being the most affected, thereafter the parietal, followed by temporal and occipital regions. In the occipital there are several normal gyri. The two outermost layers are commonly most affected, without notable distinction. The radii are involved as much as the interradiary. Throughout the layers there is a disposition for fibre-degeneration to be present in foci associated with increase in vascularity and chrome. Variations in degree of degeneration are present between gyri and parts of individual gyri. Perpendicular fibres are only moderately degenerate, with the rest of the fibres.

Sulci.—Are quite as degenerate, in respect to fibres, as the gyri, and rather fewer are normal. Vascularity and chrome are only slight.

(21) M. E.—On admission: acute mania. Causation: third attack (no cause given for previous attacks)—this one stated to be due to worry and the puerperal state. Clinical course: she passed from mania into melancholia, and gradually became stuporose. At death: melancholia with stupor. Cause of death: phthisis. Total duration: $5\frac{1}{4}$ years. Age at death: 37.

Frontal.—Tangential fibres: extremely scanty and very degenerate. Superradiary: ditto. Interradiary: worse than above. Radii: scanty, degenerate. No perpendicular fibres. Vascularity = 0, chrome +, in patches.

Summary.—Cross-layers very scanty and degenerate; interradiary the worst, and superradiary the next. Radii participate. Vascularity = 0. Chrome +.

Parietal "A."—Tangential fibres: not scanty, moder-

ately degenerate. Superradiary: ditto. Interradiary: normal. Radii: ditto. Vascularity and chrome = 0.

Summary.—Moderate degeneration outermost, two layers.

Parietal "A" (same part).—As in last. Some perpendicular fibres exist. Vascularity = 0. Chrome +, irregularly.

Temporal.—Tangential fibres: numerous, moderately degenerate. Superradiary: very scanty and degenerate; worse. Interradiary: moderately degenerate, fairly numerous. Variations in degree of degeneration in the gyrus. Radii: ditto. Perpendicular: very scanty and moderately degenerate. Vascularity = 0. Chrome +, general and in patches of intensity.

Summary.—Moderate degeneration of cross-layers and radii, with variations, superradiary excepted, these the worst. Vascularity = 0. Chrome +.

Occipital "A."—Tangential fibres: very scanty and very degenerate on summit: normal elsewhere. Superradiary: ditto. Interradiary, degenerate, but less so. Radii: trunks normal, terminals degenerate over summit. Vascularity = 0. Chrome +, worse in patches.

Summary.—Cross-layers degenerate, but only over summit. Two outermost layers worst, and terminals of radii involved.

Occipital "B" (another part).—Tangential fibres: scanty and degenerate, except on one side, where they are normal. Superradiary: more locally affected. Interradiary: very scanty and degenerate on one side, normal elsewhere. Radii: ditto. Vascularity = 0. Chrome greatly +.

Summary.—A worse specimen than last; all layers show degeneration, tangential most, others equally, with much variation in individual gyrus, normal parts adjoining diseased. Vascularity = 0. Chrome much +.

Summary of Occipital.—Cross-layers show moderate degeneration, also radii, but only in parts of the individual gyri, and some parts of the area are worse than others. No vascular increase, but chrome is +.

General Summary.—In this case of emotional disorder

the cross-layers show varying degrees of degeneration in different parts, the frontal specimens being the worst, with marked degeneration of all layers, temporal, occipital and parietal, following in order; the last has only the outermost two layers affected. In the occipital considerable variations exist within the limits of the individual gyrus. The tangential sometimes, sometimes superradiary, is worst. In the frontal specimen the interradiary is worst—an unusual condition. Radii most affected in the frontal and temporal, following cross-fibres. Vascularity is not increased with fibre-changes, whereas chrome is—an unusual condition. The perpendicular fibres, as far as noted, follow the others in point of degeneration.

(22) D. L.—On admission: delusions of persecution. Causation: heredity only assigned. Clinical course: the state persisted for some five years, then dementia became more apparent. At death: dementia with mild delusions. Total duration: 37 years. Cause of death: pneumonia.

Frontal "A."—Tangential fibres: scanty and degenerate. Superradiary: worse, very scanty and very degenerate. Interradiary: worse: extremely scanty and very degenerate. Radii: scanty and degenerate; terminals worse. Vascularity and chrome = 0. No Baillarger band.

Frontal "B" (another part).—Much as above, only tangential shows variations. Some degenerate perpendicular fibres.

Summary.—Marked degeneration of cross-layers, interradiary worst, superradiary next. Terminals of radii equally bad. Baillarger not present, and perpendicular fibres degenerate. Vascularity and chrome = 0.

Parietal "A" (2 gyri).—Tangential fibres: extremely scanty and very degenerate in one; numerous; moderately degenerate in other. Superradiary: normal in places, very degenerate at others; summit degenerate. Interradiary: normal in one, scanty and degenerate at summit in other. Radii: moderately degenerate in one, great degeneration in other. Vascularity = 0. Chrome +, only at sides.

Summary.—Cross-layers (especially two outermost) and

radii show degeneration, the latter as much as the former, with marked variations between neighbouring gyri. Vascularity = 0. Chrome +, regularly.

Parietal "B" (another part : 1 gyrus).—Tangential fibres : extremely scanty and very degenerate. Superradiary : rather less affected. Interradiary : slightly scanty and slightly degenerate. Radii normal. Vascularity and chrome as in last.

Summary of Parietal.—All cross-layers degenerate, tangential worst ; superradiary next. One specimen shows local variations, the other does not. Radii show quite unlike conditions in the two specimens. Vascularity does not follow fibre changes, but chrome is increased. One sulcus ; much same conditions as gyri ; the two outermost layers of fibres affected.

Occipital "A" (2 gyri). — Tangential fibres : fairly numerous ; moderately degenerate. Superradiary : normal ; many strong fibres. Interradiary : ditto. Radii : ditto. No vascularity. Chrome +. Some perpendicular fibres.

Summary.—A normal specimen, except for moderate tangential degeneration. One sulcus : normal.

Occipital "B" (another part : 2 gyri).—Same description as for last. Chrome is +, and general. One sulcus : normal.

Temporal "A" (2 gyri).—Tangential fibres : very scanty and very degenerate. Superradiary : worse. Interradiary : very scanty and moderately degenerate. Radii : very degenerate. Vascularity is slight. Chrome general, and also increased in patches.

Summary.—All cross-fibres and radii show considerable degeneration, the outermost layers chiefly, superradiary most. Vascularity not proportionately increased. Chrome is +. One sulcus ; same conditions as gyri.

Temporal "B" (another part).—The same description as for last. One sulcus : the same.

General Summary.—In this case of delusional insanity, with terminal dementia, the cross-layers show marked scantiness and degeneration in frontal and temporal regions to about an equal extent. The parietal region is less

affected, and the occipital is normal, except for moderate tangential degeneration. A point is that the interradiary layer is sometimes worst and sometimes the superradiary. The tangential is not oftener the worst. No local variations, except in parietal. In the two worst regions the radii show considerable degeneration, but less in the parietal. Vascularity is not increased, notwithstanding the fibre-degeneration (and that the case died from pneumonia). Chrome is generally +, and therefore does not follow vascularity. In the very degenerate frontal no Baillarger band is made out, and the perpendicular fibres are degenerate. Sulci follow gyri, showing much the same degenerations in the degenerate parts, and being normal in normal parts.

(23) H. L.—On admission: subacute mania. Causation: senility +, a previous attack (33 years before, lasted 12 months). Clinical course: the same state until death. Total duration: two months. Cause of death: chronic renal disease and brain atrophy. Age at death: 66.

Frontal "A" (7 gyri).—In six gyri, tangential fibres, superradiary and interradiary, normal, but some degeneration of fibres observed in the two latter layers about foci of vascularity and chrome. Radii: normal. Baillarger well marked. Some perpendicular fibres noted. In gyrus 7 all fibres scanty and moderately degenerate; parts of the gyrus worse than others. Vascularity + in all; chiefly superradiary and interradiary. Chrome + and around the vessels.

Summary.—Cross-layers and radii normal in six out of seven gyri, except in connection with areæ of vascularity. No. 7 is degenerate. Vascularity and chrome +.

Sulci (7).—In four, fibres are normal; in three, scanty and degenerate. Vascularity and chrome +.

Frontal "A" (same part: 8 gyri).—The same conditions as in "A."

Frontal "B" (another part: 13 gyri).—Tangential fibres normal in eleven gyri, except for moderate degeneration in connection with congested vessels. In Nos. 12 and

13 they are scanty and very degenerate; worst in 12. Superradiary: ditto. Interradiary: ditto. Radii: normal in nine, except about vessels; in two others only the terminals degenerate at summit; in 12 and 13 the radii very degenerate. Vascularity + in all, worst in 12 and 13. Chrome +, follows vessels.

Summary.—Cross-layers and radii normal in eleven gyri out of thirteen, except for moderate degeneration in connection with vascular disturbances. They are very degenerate in the two remaining gyri. Vascularity and chrome + in all, but worst in last-mentioned gyri.

Sulci.—Outer two layers moderately degenerate in all except two, where fibres are normal. Vascularity and chrome irregularly increased.

Frontal "B" (same part, 13 gyri).—The conditions are the same.

Summary of frontal region.—The cross-layers and radii are normal in the great majority of gyri, except where they abut on foci of vascularity and chrome.

In the minority of gyri the medullated fibres are either moderately or, at times, very degenerate, with occasional variations in degree in individual gyri, and vascularity and chrome are more marked in these. The sulci show moderate fibre-degeneration, with vascularity and chrome +; but many sulci are normal.

Parietal "A" (in or immediately behind ascending parietal: 7 gyri).—Tangential fibres: five gyri show degeneration, No. 1 being worst; variations in individual gyri in respect of degeneration; two gyri normal, except where fibres trench on congested vessels. Superradiary: three gyri show degeneration, to which above applies; four are normal, with similar reservations. Interradiary: two show moderate degeneration, as above; five normal, similarly. Baillarger and perpendicular fibres well marked in some. Radii: only one gyrus shows degeneration. Vascularity +; chrome + only in the most vascular gyrus, No. 7.

Summary.—The tangential fibres show the most widespread degeneration, the condition being more limited as

one proceeds from without inwards; radii only affected in one gyrus. Even in the normal gyri there are degenerate fibres about the congested vessels in all layers. Variations in intensity of degeneration, as described. Vascularity +, but chrome generally not so.

Sulci.—Fibres normal in majority, and vascularity and chrome are +.

Parietal "A^1" (same part: 8 gyri).—Tangential fibres: scanty and very degenerate in all. Superradiary: very scanty and very degenerate. Interradiary: ditto. Baillarger exists only in two. Radii: degenerate; variations in different gyri. Vascularity +; chrome = 0.

Sulci.—Fibres degenerate, and vascularity and chrome + in all, some worse than others.

Summary.—A much worse specimen than last, though taken very near it. All cross-layers very degenerate, and radii involved, though to a less degree. Vascularity +; chrome = 0.

Parietal "A^2" (same part: 6 gyri).—Same summary as in last; vascularity + in all; chrome = 0, except in one gyrus.

Sulci (9).—Fibres throughout very scanty and very degenerate, especially outermost two layers. Vascularity and chrome +.

Parietal "A^3" (same part: 8 gyri).—Tangential fibres: normal in all, except No. 8; these very degenerate, especially at summit. Superradiary: ditto. Interradiary: ditto. Baillarger band well marked; some perpendicular fibres. Radii: ditto. Vascularity + in all, except one gyrus; worse locally in individual gyri; worst in No. 8. Chrome +; worst in No. 8. Three gyri free.

Summary.—A much better specimen than last. Medullated fibres normal in all, except in one; here very degenerate, especially at summit. Vascularity +, out of proportion to fibre-changes, but most marked where latter worst. Chrome much the same, but less widespread.

Summary of parietal.—There are great variations in the state of the medullated fibres in different sections; in some sections only the tangential fibres show consistent de-

generation, in others, taken very near the last, all fibres show great degeneration in all gyri. In others, again, all fibres are normal. In respect to all otherwise normal gyri a reservation is made as regards foci of fibre-degeneration in all layers in connection with vascularity. Baillarger's band and the perpendicular fibres are found in the most normal gyri. Vascularity is +, chiefly as large, isolated, congested vessels, and follows fibre-changes. Chrome does not regularly follow vascularity, and is often absent. The sulci follow gyri approximately; those connected with better sections have normal fibres, and *vice versâ*. Vascularity and chrome are +.

Parietal posterior to ascending parietal "A" (8 gyri).—Tangential fibres: scanty and moderately degenerate in all. Superradiary: ditto; in most gyri worse than tangential. Interradiary: scanty and moderately degenerate; no Baillarger. Radii: scanty and moderately degenerate. Vascularity + greatly in foci. Chrome +, around vessels. A few degenerate, perpendicular fibres. Seldom variations in individual gyri.

Summary.—The cross-layers and radii are throughout degenerate, chiefly in moderate degree, but superradiary are the worst. Baillarger absent, perpendicular fibres degenerate. Vascularity and chrome + in foci; large blocked vessels.

Sulci.—Vascularity and chrome + in all and fibres scanty, except in one normal specimen; but vascularity is + in this also.

Parietal posterior to ascending parietal "B" (another part: 7 gyri).—Tangential fibres: scanty, moderately degenerate. Superradiary: ditto. Interradiary: slightly degenerate and scanty. Baillarger more marked in some than others. Radii: normal except in one, where moderately degenerate and scanty. Vascularity +, but chiefly capillary. Chrome is slight. Strong fibres very rare.

Summary.—A better specimen than last, the two outermost layers only being equally affected. Vascularity and chrome are also less.

Sulci.—As in gyri.

Summary of parietal posterior to ascending parietal.—The appearances differ in different parts, one being more degenerate than the other. Degeneration is usually moderate. Superradiary region in places the worst. Interradiary not always better than tangential. Baillarger band and perpendicular fibres affected in more degenerate parts, and vascularity and chrome greater.

Sulci.—These in general show less degeneration. Vascularity and chrome +.

Occipital "A" (far back in the lobe, near tip: 7 gyri). Tangential fibres: scanty and moderately degenerate in all, chiefly on summit. Gyri differ in degree, and individual gyri show differences, parts normal and others degenerate. Superradiary: ditto. Interradiary: normal. Radii: ditto. Vascularity and chrome +, in association in all. Strong fibres in fair amount in all layers.

Summary.—The two outermost layers show moderate degeneration, with variations as described. Vascularity and chrome +.

Sulci.—Fibres normal. Vascularity + in all and chrome in some.

Occipital "B" (another part, more forward: 9 gyri).—Tangential fibres and superradiary: scanty and moderately degenerate in six gyri; in rest of gyri all fibres normal. Baillarger well marked in some. Vascularity and chrome +, as in last.

Sulci (6): normal; a capillary vascularity.

Summary.—Much the same conditions as in "A."

Occipital "B" (same part: 9 gyri).—Same conditions as last.

Summary of occipital.—But little fibre-degeneration, only the outermost two layers showing it in moderate and equal degree. Variations in degeneration in parts as described. Vascularity and chrome + in association. Baillarger fibres and strong, are not affected. The sulci are less affected, vascularity only being moderately increased; no fibre-changes.

Temporal "A" (4 gyri).—Three normal. In fourth: tangential fibres: moderately degenerate. Superradiary:

ditto. Interradiary: ditto. Radii: extremely degenerate. Vascularity + in all, chiefly interradiary, worst in fourth. Chrome + in all, but less in fourth.

Summary.—Normal in greater part except for increase in vascularity and chrome. One gyrus shows moderate degeneration of cross-layers and much greater of radii. Vascularity and chrome +, though former alone follows fibre-degeneration.

Sulci (3).—Superradiary: fibres degenerate in one, and vascularity and chrome +.

Temporal "A^1" (same part, 10 gyri).—Tangential fibres: six are normal and four others also except for scantiness and moderate degeneration at summit. Superradiary: ditto. Interradiary: nine normal, one very degenerate. Baillarger's band in many of the normal. Radii: normal in eight, and in other two only terminals degenerate. Vascularity and chrome + in all, chiefly interradiary.

Summary.—Cross-fibres and radii normal in the main, the outermost two layers showing moderate degeneration at summit in a minority. One very degenerate gyrus as regards interradiary. Vascularity and chrome +.

Sulci (7).—Normal except for moderate degeneration in connection with vascular patches.

Temporal "A^2" (same part: 4 gyri).—Tangential fibres: scanty and degenerate and worst at summit in three, normal in one. Superradiary: ditto. Interradiary: normal. Radii: normal in two, in others scanty and moderately degenerate, especially at summit. Vascularity and chrome +, chiefly interradiary.

Summary.—The two outermost cross-layers moderately degenerate, with local variation, in majority of gyri. Interradiary: normal, whilst radii moderately involved in some gyri. Vascularity and chrome +.

Sulci (3).—Fibres normal. Vascularity and chrome +, with variations between sulci.

Summary of temporal "A."—Moderate degeneration of cross-layers, especially outermost two, in minority of gyri. Variations in degree of degeneration in individual gyri frequent. Radii: less affected, but to a greater extent than

cross-fibres in one gyrus; one very degenerate interradiary layer also seen. Baillarger's band in many of them normal. Vascularity and chrome + uniformly, and in association. Sulci show also only moderate degeneration of fibres in connection with patches of vascularity and chrome.

Temporal "B" (another part, 6 gyri).—Tangential fibres: normal in five, scanty and moderately degenerate over summit in sixth. Superradiary: three normal, others scanty and moderately degenerate. Interradiary: normal, except one, where the fibres moderately degenerate over summit. Baillarger hardly exists. Radii: fibres and terminals degenerate, especially at summit in Nos. 1 and 2; four are normal. Vascularity and chrome + in all, with variations in different and in individual gyri, chiefly interradiary.

Summary.—Cross-fibres not free from moderate degeneration, though this is very limited, most gyri being normal. The superradiary most widely degenerate. Interradiary not better than tangential, and radii are as much involved as either. Vascularity and chrome +, rather out of proportion to fibre-changes.

Sulci (5).—Fibres chiefly degenerate. Vascularity and chrome + in all with variations.

Temporal "B" (same part, 9 gyri).—In seven, tangential fibres are normal; moderately degenerate and scanty, chiefly at summit in two. Superradiary: normal in all. Interradiary: ditto. Radii: ditto. Perpendicular fibres fairly numerous and normal. Vascularity and chrome + in all, chiefly interradiary.

Summary.—Normal conditions with slight or very localised tangential degeneration in two. Vascularity and chrome also disproportionately increased.

Sulci (7).—Fibres normal, but vascularity and chrome greatly +.

Summary of temporal "B."—Very limited and moderate degeneration of cross-fibres and of radii. The gyri are chiefly normal. One specimen rather worse than the other, although cut from very near latter. Perpendicular fibres normal. Vascularity and chrome +, and out of proportion to fibre-changes.

Sulci.—These agree with the gyri in presenting moderate fibre-degeneration, not widespread, and vascularity and chrome +, out of proportion to this.

Summary of temporal.—The cross-layers are only moderately and very locally degenerated, the majority of the gyri being normal. The radii on the whole are less affected, but in rare instances as much and even more. Variations in degree of degeneration in individual gyri: often only a very limited part involved, rest of gyrus normal. Baillarger's band and perpendicular fibres noted in many of the normal. Vascularity and chrome + generally and often out of proportion, as stated.

Sulci.—Only moderate degeneration, where affected: largely normal. Vascularity and chrome +, general.

General summary.—In this case of subacute mania the degeneration of the cross-layers and radii is very seldom striking, a large number of gyri being normal throughout the hemisphere, and only moderate degeneration, as a rule, occurring where any is present. Throughout there is an increase in the fibre-degeneration locally in connection with patches of vascularity and chrome. Here the impression left is that the vascular changes were initial and those of the fibres secondary: the vascular patches are often very prominent, composed of large blocked vessels, whilst the fibre-degeneration is, as a rule, moderate. And outside these foci the fibres are so frequently normal; and the general vascularity is often out of proportion to the changes in fibres. Some very degenerate sections are in close proximity to others nearly normal. In one region (hind-parietal) it is noted that the superradiary layer is often the worst: otherwise the two outermost layers are on the whole the most affected, though superradiary is as much involved as tangential, and fairly often the interradiary layer is as involved as either.

Variations in degree of degeneration occur frequently between gyri and parts of individual gyri. Baillarger, strong, and perpendicular fibres are noted as well marked where gyri are normal, and absent or degenerate and scanty in some of the worst specimens. In the frontal, central, and

parietal regions the radii show moderate degeneration in several gyri, the terminals being worse; less in temporal region. In the occipital they are normal. Vascularity and chrome are generally +, and go together with fibre degeneration, though vascularity is in some areæ + out of proportion thereto. Foci of large, blocked vessels, with deep chrome-areæ about them, occur. There is no marked difference in degree of degeneration between the different parts of the hemisphere: the central and paretial regions are rather the worst, and frontal is no worse than occipital and temporal; the latter three about equally involved.

Sulci.—Are chiefly like the gyri, showing normal condition of fibres usually, and only moderate degeneration where any. Vascularity and chrome are +, and out of proportion to fibre-changes.

(24) R. M.—On admission: acute mania; much incoherence; hallucinations; no violence. No cause known (a vagrant). Clinical course: became quieter, but remained hallucinated and deluded and very incoherent. Able to work. This was the condition at death, which was due to pneumonia. Age at death: 27. Total duration: 5—6 years.

Pre-central (9 gyri).—Tangential fibres: very scanty and very degenerate. Some variation in gyri; in the worst they are practically absent. Superradiary: ditto. Interradiary: less scanty, but almost as degenerate; no Baillarger. Radii: very scanty and very degenerate. Variations between gyri, in respect of these and last. Vascularity +, generally, chiefly capillary. Chrome +, but less regularly, and not always following vascularity. In No. 9 chrome obliterates all fibres, except tangential. No perpendicular and no strong fibres.

Summary.—Great degeneration of cross-layers of radii. Tangential and superradiary worst and equally affected. Occasional variations between gyri. Vascularity +, diffuse. Chrome +, but less regularly, and not always with vascularity. In great excess in one gyrus.

Pre-central "A^2" (9 gyri, same part).—The same conditions.

Sulci (9).—Fibres very scanty and very degenerate on the whole, with fewer variations than on gyri. Vascularity and chrome + and together.

Parietal "A" (2 gyri).—Tangential fibres: abundant, moderately degenerate. Superradiary: scanty, moderately degenerate. Interradiary: normal. Good Baillarger. Radii: normal, terminals moderately degenerate. Perpendicular fibres: very scanty and moderately degenerate. Strong fibres in various layers. Vascularity +, large blocked. Chrome +.

Summary.—Moderate degeneration of the outermost layers, in which terminations of radii involved.

Parietal "B" (another part; 2 gyri).—Tangential fibres: very scanty and very degenerate; worse in one gyrus. Superradiary: ditto. Interradiary: very scanty and extremely degenerate in one gyrus, and Baillarger absent; the other less affected but still scanty and degenerate. Radii: in both degenerate; terminals worse. Vascularity + in both. Chrome + in both. Both less in one gyrus. Perpendicular fibres scanty; strong fibres not marked.

Summary.—Great degeneration of all cross-layers, interradiary equally with others, with variations between gyri. Radii involved, but less so, except terminals. Vascularity and chrome +, with variations as above.

Summary of parietal.—Great variation in degree of degeneration of fibres; moderate of two outermost layers (with terminals of radii) in one part; extreme of all layers, with radii involved, in the other. Strong, perpendicular, and Baillarger fibres better in the better specimen. Vascularity and chrome general, and +.

Temporal (4 gyri).—Tangential fibres: extremely scanty and very degenerate in three, less in fourth. Superradiary: less scanty, with variations in different gyri; very degenerate; summit worst. Interradiary: numerous, very degenerate. Baillarger well marked. Radii: moderately degenerate, terminals worse. Vascularity + in all. Chrome = 0 in two, except about vessels; dense in two. Strong fibres, few, degenerate; perpendicular fibres = 0.

Summary.—All cross-layers show much degeneration, progressively less from without inward; radii less affected, but terminals equally with cross-fibres. Majority of gyri equally affected. Vascularity + uniformly; chrome irregularly +.

Occipital "A" (8 gyri).—Tangential fibres: very scanty in five, numerous in three; very degenerate in all save seven and eight, these only slightly so. Superradiary: scanty in six, numerous in two, and very degenerate; only slightly so in seven and eight. Interradiary: scanty and moderately in three, normal in three (including Nos. 7 and 8), numerous and moderately degenerate in two. Baillarger only present in "numerous" ones. Radii: moderately degenerate, terminals worse; in Nos. 7 and 8 only terminals affected. Vascularity greatly + in all; chrome only slight. Perpendicular fibres: few and degenerate.

Occipital "B" (another part, 7 gyri).—The same conditions.

Summary of occipital.—The outermost two cross-layers chiefly affected, usually scanty, and commonly very degenerate in equal degree. Terminals of radii equally involved; other fibres in less degree. Two gyri much better than others. Vascularity +; chrome only slight.

Sulci (4).—Fibres degenerate. Vascularity and chrome as above.

General summary.—In this case of acute passing into chronic mania there is considerable degeneration of the cross-fibres, especially of the two outermost layers, and in equal degree in all regions, the frontal being the worst, the parietal and temporal about equally involved, and the occipital least. The radii are almost as affected as the cross-fibres in the frontal, but less in the others, though their terminals are always involved. Strong and perpendicular fibres are absent in the worst specimens. Variations between gyri and in individual gyri in the less degenerate specimens. Vascularity is consistently +, and is probably in association with the pneumonia, which caused death; but chrome does not regularly follow.

The sulci show much the same conditions as the gyri.

(25) M. B.—On admission: acute mania. Causation: no assigned cause, the condition came on as simple mania, and this produrmal state lasted 2 years. Clinical course: the simple mania passed into an acute condition which lasted 2 months, thereafter acute delirium supervened, lasting 6 weeks, about. Great emaciation; sordes; syncopal attacks. She died in one of these. Cause of death: exhaustion from acute mania: Brown atrophy of the heart. Age at death: 31.

Frontal (3 gyri).—Outermost zone is occupied by chrome and capillaries, obliterating the tangential fibres. All cross-fibres extremely scanty and extremely degenerate. The radii are also very scanty and very degenerate, scarcely less so than the others. Vascularity + general but not marked. Chrome is moderate, except in anterior zone. No perpendicular, no Baillarger, and no strong fibres.

Summary.—An exceedingly degenerate specimen.

Parietal (2 gyri).—Tangential fibres obscured by chrome and capillaries. Superradiary: very scanty and very degenerate in one, not so far gone in the other. Interradiary: scanty: some largeness: very degenerate. Radii very scanty and very degenerate. Vascularity and chrome = 0, except in outermost layer. No Baillarger fibres; perpendicular fibres rare and degenerate.

Summary.—A very degenerate specimen, not quite so bad as frontal.

Occipital (3 gyri).—The fibres are scanty and degenerate throughout, including the radii. No Baillarger band. Some perpendicular and strong fibres exist, but are degenerate. Vascularity = 0. Chrome = intense in outermost zone, and large patch occurs on one side of gyri, tending to obscure all layers.

Summary.—A very degenerate specimen, rather less so than the two preceding.

Temporal (3 gyri).—Tangential fibres: a very few degenerate to be seen, most of the zone obscured by chrome. Superradiary: scanty, but less so than in previous; extremely degenerate. Interradiary: ditto. Radii: ditto. Some degenerate, strong and perpendicular fibres seen

throughout. Vascularity = 0. Chrome slight, except for outermost layer.

Summary.—A very degenerate specimen, but on the whole the least of all.

General summary.—In this case of acute mania passing into acute delirious mania the cross-layers and radii show very great degeneration in all parts examined, the frontal gyri being the most affected, the temporal least. The tangential fibres cannot be made out, except very rarely, owing to deposit of chrome and to capillary engorgement in the outermost zone. The pia only moderately thickened. The other cross-fibres are about equally affected, and the radii are but little less involved, the degeneration in all fibres being extreme. The perpendicular fibres, the strong and the Baillarger disappear in the worst gyri, and are very scanty and degenerate in the others. Notwithstanding fibre-degeneration the vascularity and the chrome—apart from the outermost zone—are very slight.

The conditions as to *vascularity* and *chrome-deposit* in the various cases examined may be summarised as follows:—In 13 out of the 25 cases—and in various forms of insanity, mostly those of some years' duration—the vascularity is considerably increased, commensurately with the degree of fibre-degeneration. The increase is chiefly in the interradiary and superradiary layers. It is most marked in the cases of paralytic dementia (although these are of comparatively short duration). In 4 of the 13 cases there was a bodily disease, causing death (pneumonia 3, Landry's paralysis, 1), which might have been regarded as responsible for the cortical congestion; but in two other cases in which pneumonia caused death there was no such congestion. And in other cases dead of phthisis the vascularity was increased, though anæmia might have been looked for. Therefore it is fair to assume that the vascular congestion in the above-mentioned four cases was connected with cortical disease, as manifested by the fibre-changes.

In four cases in which the fibre-degeneration was considerable or extreme, the mental disorder having lasted in some a few weeks, in others several years, vascularity is only

slight, not commensurate with nerve-fibre changes. In seven others, under similar conditions, it is even entirely absent. These eleven cases embrace instances of acute and chronic insanity. In one case of subacute mania (two months' duration, death from chronic renal changes), without marked fibre-degeneration, the vascularity and chrome-deposit are increased out of proportion to latter.

Chrome-deposit (often exceedingly deep) is generally associated with increased vascularity, but does not follow fibre-degeneration quite so closely. [This deposit in the specimens must be associated with a morbid change in the tissue. The most diseased gyri show it most. Its presence cannot be explained by faulty technique.]

General Conclusions.

(1) In seventeen out of twenty-five cases marked and widespread degeneration of the medullated fibres of the cortex was observed. In six of these this was extreme, as follows:—One case of delusional insanity (6 years' duration); one dementia paralytica, maniacal type (2 years); one ditto ($5\frac{1}{2}$ months); one puerperal insanity, with variations in emotional state ($5\frac{1}{4}$ years); one acute mania, passing into acute delirium (4 months); one acute mania ($1\frac{3}{4}$ years).

In three others of the twenty-five cases there was considerable degeneration, but more locally; slighter degeneration in general. The remaining five cases showed moderate degeneration.

In some cases of paralytic dementia there was great variation in degree of degeneration between sections cut very near each other in the same piece (whole piece not above 8-9 mm. in depth).

(2) Atrophy of fibres can be more extensive and intense in cases of a few months than in those of many years' duration.

(3) The pre-central region (frontal lobe) showed the most degeneration in nine out of twenty-three cases (in two remaining it was not examined), and was equal to the worst region in four others. In only one case was this the least

affected area. Next followed the temporal region; worst in four, equal to worst in two. The centro-parietal region is least affected in eight, the majority of instances, and is worst only in one case.

The fibre-degeneration is often quite as marked in other parts as in the frontal region, and sometimes more so.

The occipital lobe is not spared in general paralysis of the insane as Tuczek stated; this statement Zacher has already denied.

(4) As regards the cross-layers: the tangential and superradiary are the worst and earliest involved, and are equally affected in the majority of cases. The superradiary rather than tangential is oftener the worst. As the latest development of the "primary" fibre-deposit, the superradiary might be expected to be earliest involved. In three cases only are the three layers equally affected. The interradiary is commonly less involved, seldom as much, very rarely is worse. These conclusions are at variance with Tuczek's findings for general paralysis of the insane.

(5) The radiations are less affected than the cross-fibres in eleven cases, equally, or nearly so, in eleven, and equally locally (in certain parts) in three. They are never worse than the cross-fibres. The terminals of the radii are always worse than the trunks, where the radii are involved, and are often involved where the trunks are normal. They share equally in the degeneration of the superradiary fibres.

(6) Variations in degree of degeneration between neighbouring gyri, and also between parts of individual gyri, are frequent in the less diseased parts.

(7) The "strong" fibres and the "perpendicular" ones are very generally very degenerate or absent in specimens showing disease, whilst still degeneration is only moderate in the bulk of the cross-fibres. It is interesting to recall in this connection that the former are regarded as a later development than the latter.

(8) There is considerable increase in vascularity (congestion: sometimes more than this) in thirteen cases, commensurately with fibre-degeneration, and slight increase (non-commensurate) in four others. The increase is most

marked in the cases of paralytic dementia. Absence of vascularity in a few acute cases with great degeneration of fibres is notable.

(9) Chrome-deposit is often extreme, especially in the most diseased gyri, and generally in association with increased vascularity (about the vessels); but does not follow fibre-degeneration quite so closely as does vascularity.

(10) The fibre-degeneration in the sulci is, on the whole, equivalent to that in the adjoining gyri, as far as the outermost two layers are concerned. The interradiary fibres but seldom show disease. Vascularity and chrome are commonly similar to the conditions obtaining in the adjoining gyri.

DESCRIPTION OF FIGURES.

FIG. 1.—Normal medullated fibres. × 25.

FIG. 2.—Parietal cortex from a case of acute puerperal mania. Normal, except for slight degeneration of superradiary layer (not seen under this magnification). × 35.

FIG. 3.—To show general paucity of the cross-fibres. From a case of paranoia. × 35.

FIG. 4.—Sclerosis of outermost cortical layers (aniline blue black, fresh section). For comparison with fig. 5. × 65.

FIG. 5.—A section from same part as fig. 4 (corresponding). Showing persistence of tangential fibres in fair number notwithstanding the sclerosis of outermost zone shown by the fresh method in fig. 4. Chronic mania. × 65.

FIG. 6.—Showing scantiness and degeneration of the radii and interradiary fibres, especially varicosities. Chronic melancholia; dementia. × 300.

FIG. 7. Showing great paucity of all fibres, vascularity and chrome-infiltration, and patches of sclerosis in outermost zone. Acute melancholia. × 65.

FIG. 8.—Destruction of medullated fibres, vascularity, chrome-infiltration. Paralytic dementia. × 25.

FIG. 9.—Same conditions as last. Same case. × 25.

FIG. 10.—Tortuosity of the radiations. Paralytic dementia. × 300.

FIG. 11.—Foci of vascularity and chrome. The fibres only moderately diseased. × 35 (but somewhat higher magnification required to show the superradiary). Subacute mania.

FIG. 12.—Extreme destruction of fibres, including radiations; fibres almost entirely absent. Greatly increased vascularity and chrome-infiltration. Paralytic dementia. × 35.

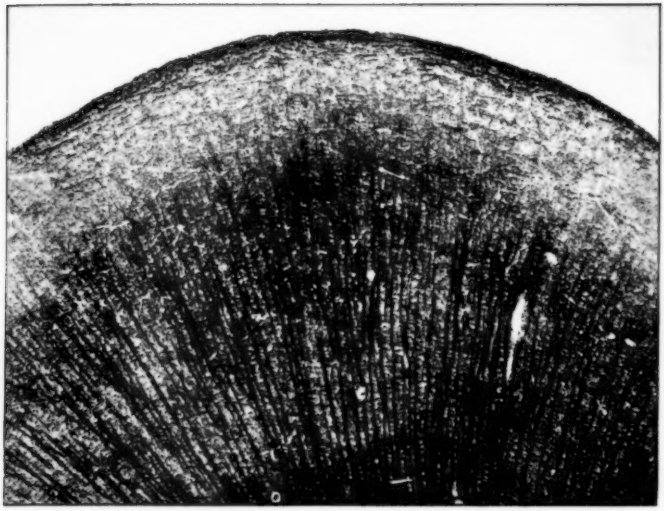

FIG. 1.

FIG. 2.

FIG. 3.

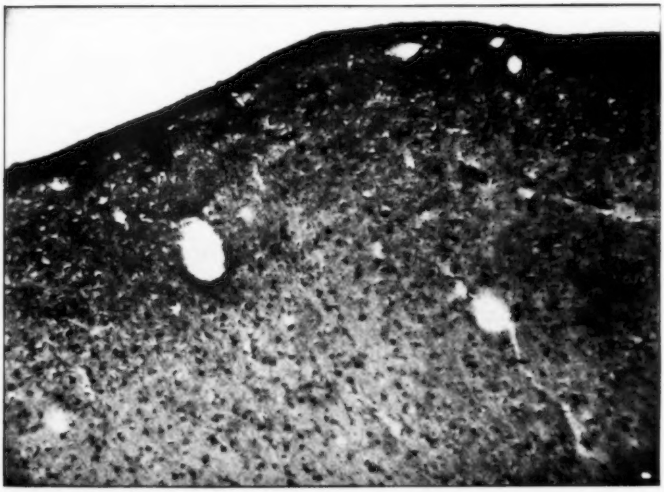

FIG. 4.

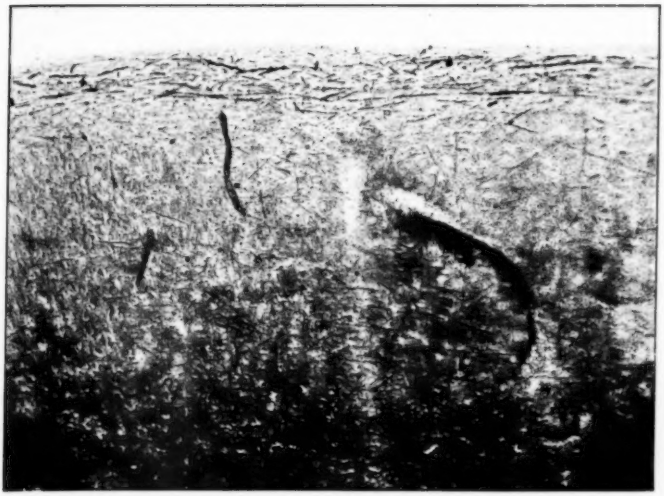

FIG. 5.

FIG. 6.

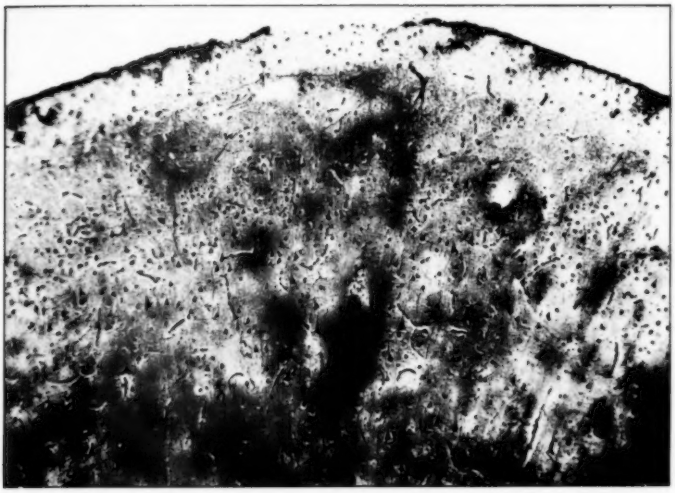

FIG. 7.

FIG. 8

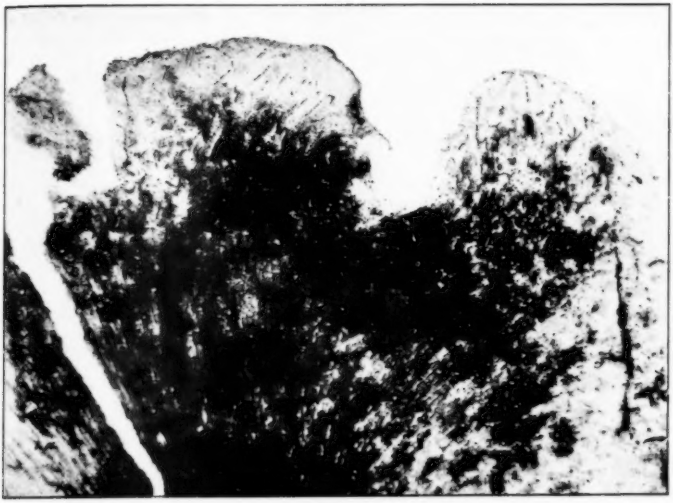

FIG. 9.

FIG. 10.

Fig. 11.

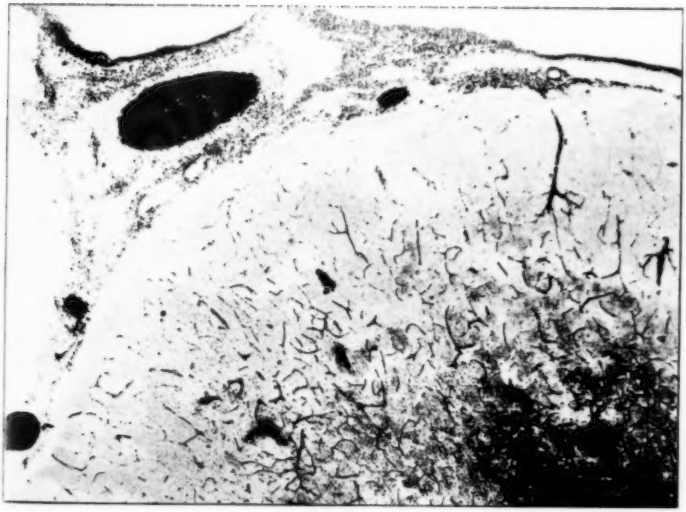

Fig. 12.

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OBSERVATIONS ON THE COURSE OF THE OPTIC FIBRES IN A CASE OF UNILATERAL OPTIC ATROPHY.

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THAT in man a semi-decussation of the optic nerves occurs at the chiasma is almost universally believed; the fact, however, that the veteran histologist Kölliker disputes this position has given a fresh impetus to observations on the question.

Kölliker's position, as described in the last edition of the *Handbuch der Gewebelehre*, is that pure anatomy yields evidence of a complete crossing in man, in the dog and the cat.

His observations were made by serial horizontal sections through the chiasma stained by the Weigert method.

In order to study the course of the fibres a complete series must be made; this is not easy and Kölliker criticises Gudden's original pictures on the ground that they are merely schematic, and that the distinct uncrossed bundle there figured can never be found in nature.

Kölliker (1) has again only last year returned to the subject, and has studied, by means of the Golgi method, the course of fibres in the chiasma of sheep, cats and man. The results he has obtained are that in man it is impossible that any considerable number of fibres pass to the tract of the same side as the optic nerve from which they are derived.

though he admits the existence of a very small number of direct fibres.

In addition to this conclusion Kölliker has noticed the important fact that in the cow, cat, rabbit and dog, a division of individual fibres takes place in such a manner that a fibre from one optic nerve passes to the two tracts.

In man Kölliker was only able to find in his specimens one such fibre.

Should further observations demonstrate that division of fibres is a constant occurrence in the chiasma, Kölliker considers this fact would remove in considerable part the physiological objections to his view.

Finally Kölliker urges that the minute anatomy of the chiasma must be more thoroughly observed before the question is settled.

Other authors also favour the view that the decussation is complete, viz., Biesiadecki (2) in 1861, Mandelstamm (3) 1873, Scheel, W. Krause, 1897, and especially Michel (4) 1873-1890.

Mickel maintains:—

(1) That serial sections of the chiasma give no support to the view of semi-decussation.

(2) That investigations of degenerations in man and animals give no evidence of a non-crossed bundle of fibres.

(3) "He argues that if homonymous bilateral hemianopsia is to be explained by the decussation in the chiasma, the proportion of crossed to uncrossed fibres should be as 1·1, and contrasts with this the admission of even those who adhere to the doctrine of partial decussation, that the relation is 3·1, or 4·1, at most as 5·3." (Quoted from Barker, "The Nervous System.")

Our case does not, however, show any marked difference between the amount of degeneration in the two tracts. Further, owing to the limitation of the field of vision, the outer and lower parts of the retina are probably less developed than the inner and lower, and, as Michel himself has found, the number of fibres passing to different sections of the retina is very variable, being least in the outer segment.

Michel also cites the fact that no anatomical evidence has yet been produced demonstrating the double representation of the macula, which is required by the theory of semi-decussation in order to explain the freedom of the fixation point in cases of hemiopia. Michel's hypothesis cannot, however, be regarded as giving a more satisfactory explanation.

Finally Kölliker has been attacked on his own ground by Grützner (5) who, in an admirable critical article, concludes that anatomy can give no definite reply to the question of the complete or partial decussation of the optic fibres. This author made models of the chiasma, in which one half of the fibres were made to cross, the other half to remain on the same side. The models were then embedded in paraffin and cut into a series of horizontal sections. From a study of these sections he states that if he had not been in possession of the actual facts, he would have judged that *all* the fibres were crossed.

In view of the renewed interest which the subject has thus recently attracted we thought it permissible to bring forward the following case of unilateral atrophy of an optic nerve, which shows clearly the existence and arrangement of a semi-decussation in the chiasma, and furnishes also some other points of interest.

Résumé of Case.

Sarah B., aged 63, admitted to the Liverpool Royal Infirmary on January 14, 1898, under the care of Dr. Caton, to whom we are indebted for permission to record the case. Three years previously the patient had received an injury to the right eye, and six months before admission enucleation became necessary and was performed at the Stanley Hospital. On admission to the Royal Infirmary the patient was found to have a greatly enlarged liver. Death occurred in about three weeks after admission, and the autopsy revealed the presence of a large melanotic growth in the liver.

We record here :—

(1) The resulting degenerations in the nerve, chiasma and optic tracts.

(2) The existence of undegenerated fibres in the atrophied nerve.

(3) The condition of the oculomotorius nucleus.

Method of Examination.

The whole brain was hardened in Müller's fluid, the optic system, with the neighbouring parts of the brain undisturbed, was then removed. By this means it was easy to localise the position of any degenerations that might be found.

The optic chiasma with pieces of each tract and both nerves was removed and placed in Marchi's fluid. The remaining extent of the tracts, with the adjoining brain tissue, including the geniculate bodies and pulvinar, was cut into small sections, 3 mm. in thickness, transverse to the long axis of the tract fibres. These, together with separate pieces of both optic nerves, were also placed in Marchi fluid. The tissues were afterwards embedded in paraffin, cut in serial sections of 10 μ each and then fixed on slides. The chiasma was cut into a complete series of horizontal sections, the remaining parts were cut transversely. As no fibres showed the characteristic Marchi globules they were stained with the acid hæmatoxylin of Schäfer and then treated with Pal's fluids.

This method of paraffin embedding has the advantages that a complete series may be cut, and that the sections are much thinner than can be obtained by the celloidin method. The previous treatment of the tissues by the Marchi fluid enables the hæmotoxylin to stain perfectly well.

I.—*The Resulting Degenerations of the Nerve, Chiasma, and Tracts.*

The naked eye examination of the sections showed that the right optic nerve was completely degenerated, being white and shrunken.

The optic tracts were plainly seen to contain sclerosed tissue, which stood out markedly in contrast to the black-stained nerve-fibres.

Optic nerves.—The left nerve showed the normal structure. The right practically consisted of fibrous tissue; it was very much shrunken and complete atrophy appeared to have taken place.

Careful examination, however, with a $\frac{1}{12}$ in. immersion lens, discovered a few normal nerve-fibres, scattered irregularly over the area of the section. In diameter they never measured more than 1·5 μ, and hence belonged to the smallest of the optic nerve-fibres (see plate, fig. 1).

Chiasma.—The chiasma, with the optic nerves attached as described, was cut in a complete series of about 500 horizontal sections, and every fourth one stained. By means of this large number of fairly thin sections, the course of the degenerated tracts could be fairly well traced, and is shown in the accompanying diagrams (figs. 1—5). In the sections about the median plane (figs. 3 and 4 and plate, fig. 4) the atrophy from the right nerve is seen spreading in the central portion of the same-sided tract, and occupying the front and back of the chiasma—its extent being larger in the latter situation.

As the sections approached the upper and lower surfaces the distribution of atrophied tissue was less defined, and the intermingling of normal fibres was greater. The general relations were, however, the same. In looking at figs. 3 and 4 it seems difficult to resist the conclusion that the atrophied tract at B corresponds with the normal fibres at A. The general "grain" of the fibres of the left nerve appear to be sweeping into the left tract, much as the atrophic tissue of the right nerve passes out of the right tract.

Is it possible, despite the observations of Grützner, to rely upon the anatomical evidence?

When we examined this region (*A*) in very thin sections (6 μ) with ocular 4 Zeiss, and $\frac{1}{12}$th inch objective, large strands of fibres cut in longitudinal direction were certainly seen passing in two directions, viz.: (1) streaming through the chiasma; (2) curling round to enter the tract of the same side.

The sections were so thin that the curve on groups of

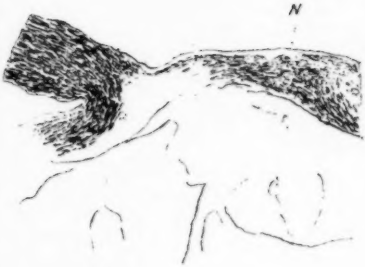


FIG. 1.

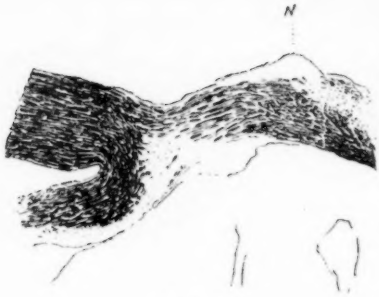
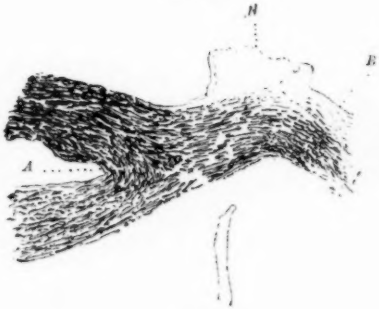


FIG. 2.

FIG. 3.



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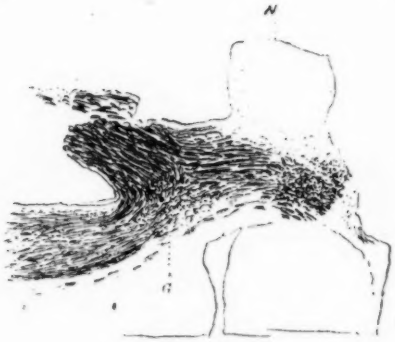

FIG. 5.

FIGS. 1 TO 5.

Series of drawings of horizontal sections of chiasma, made by Edinger's apparatus. Fig. 1 is the most dorsal, fig. 5 the most ventral section.

N = Atrophied right optic nerve. *G* = Gudden's commissure.

A = Junction of left nerve with left tract. *B* = Junction of right nerve with right tract.

individual fibres could be distinctly seen, and we are inclined to think that the appearance represented an anatomical fact. As pointed out by Michel, the fibres of the optic nerve, as they enter the chiasma, turn sharply at right angles, and again at another right angle when entering the tract of the opposite side. It is this fact which is the cause of the deceptive appearance in anatomical preparation, inclining the observer to believe the fibres enter the tract from the nerve of the same side.

FIG. 6.

Bearing in mind this warning, we are yet of opinion that is not sufficient to explain the appearances alluded to. In these very thin sections groups of fibres having the same direction can be traced along the outer border of the nerve and into the tract of the same side.

Right optic tract.—Sections taken near the chiasma showed that the atrophy occupied a central position extending further outwards than inwards (figs. 6, 7, 8).

Microscopical examination revealed that in no part was there complete atrophy, normal nerve fibres being found in the most affected part of the tract. Many of the normal fibres found in the atrophic tissue were of small diameter (1·5—2 μ), only the minority being as large in diameter as 5—7·5 μ: whereas at the periphery of the section these large fibres greatly preponderated (plate, figs. 2 and 3).

FIG. 7.

The same general distribution was noted in the sections taken from the more centrally situated part of the tract, but the extent of the atrophy gradually diminished.

At the commencement of the level of the external geniculate body no obvious degeneration was seen, but microscopically the fibres on the outer part of the tract

FIG. 8.

FIGS. 6 TO 8.

Series of drawings of transverse sections through the right optic tract. Fig. 6 is nearest the chiasma, fig. 8 near the geniculate body.

FIG. 9.

FIG. 10.

FIG. 11.

FIGS. 9 TO 11.

Series of drawings of transverse sections through the left optic tract. Fig. 9 is nearest the chiasma, fig. 11 near the geniculate body.

were considerably larger and more numerous than those on its inner aspect. Sections through the geniculate body showed that its cellular mass was richly covered by fibres of a large diameter; those deeper in its substance were much smaller.

Left optic tract.—In sections near the chiasma the degeneration is well seen, situated peripherally and chiefly on the inner and inferior aspect of the tract. It is never complete, normal nerve-fibres being present in the connective tissue. Its extent gradually diminishes, as it is traced centrally (figs. 9, 10, 11).

Near the geniculate body the area of degeneration could no longer be observed with the naked eye, but on microscopic examination, the inferior region was found scantily supplied with nerve-fibres.

No obvious difference in size was found between those normal fibres situated in the atrophied region and those composing the undegenerated portion of the optic tract.

Remarks.

Those who, on anatomical grounds, oppose the prevalent view of a semi-decussation of the optic nerve-fibres, must explain the degeneration appearances in cases similar to ours.

Michel (6) appears to be the only observer who, using the modern methods, has given cases of degeneration and maintained that these support the view of a complete decussation. He mentions and illustrates four such cases which, however, do not appear to us to justify his position; for if, as his figures show, there is no degeneration visible in the tract on the same side as the atrophy—there is only *partial* atrophy in the opposite tract—indeed, as Wieting has remarked, the unprejudiced observer would draw the opposite conclusion to that which Michel does from a study of his figures. The very old observations of Biesiadecki, the more recent ones of Mandelstamm, were made on unstained preparations, and cannot well be compared with Weigert-Pal specimens.

On the other hand, Purtscher (7) records six cases, Wieting (8) three, Siemerling (9) and Williamson (10) one case, stained by the Weigert method, which clearly show the atrophy in both tracts, and in a similar position to that described by us, viz., the crossed fibres occupy the centre of the tract, the uncrossed the periphery. Marchand (11) and Burdach (12) give figures showing clearly atrophy in both tracts, but having a different position.

Hellendall (13) quotes three well illustrated cases showing that the crossed bundle occupied an inner and ventral position in the tract, whilst a triangular field of atrophied tissue was situated just at the commencement of the same-sided tract, which could not, however, be traced any further.

Hellendall and also Hausemann (14), who criticised the cases, considered that the preparation showed also *anatomical* evidence of the existence of uncrossed fibres in the tract on the same side as the normal nerve.

Thus summarising we may state :—

(1) That all observers with the modern methods, with the single exception of Michel, are of opinion that degenerations occur in both tracts after destruction of one optic nerve.

(2) That whilst in general pure *anatomy* cannot demonstrate a decussation, certain cases, *e.g.*, Hellendall's and the present writers', yield some evidence of an uncrossed bundle of fibres.

(3) That the commonest position in which such degenerations in the tracts have been found corresponds to that described above.

II.—*On the Existence of Undegenerated Fibres in the Atrophied Nerve.*

We alluded to the presence of very fine normally-stained fibres in the stump of the atrophied nerve, and to the fact that the fibres remaining in the degenerated portion of the tract of the same side also had an extremely small diameter.

Gudden long ago asserted that the fine fibres of the optic nerve were connected with the anterior corpus quadrigeminum, whilst those of larger diameter could be traced

to the geniculate body. Michel very clearly figures similar fibres, Williamson and Wieting also found them, and the latter observer suggested they might be centrifugal in their course.

Hitherto the existence of centrifugal fibres in the optic nerve has been demonstrated by the Golgi method, and their origin could not be ascertained in this way. Recently, however, Wallenberg (15), Boyce and Warrington (16) showed that in the bird a lesion of the optic lobes gave rise to a degeneration extending to the opposite retina. These various observations certainly suggest that the fine fibres may belong to centrifugal neurons, though the evidence adduced is not sufficient to show where their cell origin is.

As shown in the photograph (plate, figs. 2 and 3) the small size of the fibres in the degenerated centre of the tract in comparison with those of the normal periphery is very noticeable, and suggests the possibility that they may belong to the same system as those found undegenerated in the atrophied stump, viz., have a centrifugal course.

III.—*On the Condition of the Oculomotorius Nucleus.*

A series of paraffin sections was made, including the whole of this nucleus, and every fifth section stained by Held's modification of the Nissl method. It is now well known that section of an axis cylinder is followed in general after a short time by the change known as chromatolysis in its nerve cell. Further, that after a considerable period the cells so affected tend gradually to return to their normal condition. In our case the cells of the nucleus were throughout *normal*. It is possible that this repair may have taken place, as observation by Van Gehuchten, Marinesco, and one of us have shown that after a period of 100 days not any very obvious difference is noticeable in any group of cells whose axons have been cut. The application of the law of Nissl for the limitation of a nucleus of origin is not always very satisfactory. Bernheimer (17) and Schwabe (18) have made laborious researches on the connections of the various parts of the oculomotorius nucleus, but their results are

not in close agreement. Thus Bernheimer considers that the external muscles of the eye receive in the main a crossed innervation from the nucleus, whilst Schwabe regards the nerve to the superior rectus as alone derived from the opposite side of the nucleus. This author also was unable by this method to locate the nucleus of the internal eye muscles, whilst Bernheimer refers to the cells situated on the lateral part of the nucleus as giving origin to the nerve-fibres which supply these muscles.

In experiments performed by one of us (19), in which the third cranial nerve was divided within the cranium and the oculomotorius nucleus subsequently examined, only changes of a slight nature were found, which were useless for purposes of careful localisation. It is probable that in the case of the eye muscle our efforts should be directed to the localised of cell groups representing combinations rather than single movements.

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(14) Hausemann. *Ibid.*
(15) Wallenberg. *Neurolog. Centrdlb.*, 1898.
(16) Boyce and Warrington. *Phil. Trans. R. S.*, B., vol. 191.
(17) Bernheimer. *Wien. Klin. Wochen.*, 1896.
(18) Schwabe. *Neur. Centralb.*, 1896.
(19) Warrington. *Journ. of Phys.*, xxiii., 1898.

The above references are fairly complete as regards cases which have occurred in the human subject. The literature dealing with the general subject is extremely large and is to be found in the works of Gudden, Michel and v. Monakow.

DESCRIPTION OF PLATES.

Photographs taken from sections stained by Schäfer's acid hæmatoxylin method. The black masses represent osmic stain which was not removed by washing. Black rings are normal nerve-fibres.

FIG. 1.

Right optic nerve showing a few undegenerated nerve-fibres in the sclerosed tissue. (Zeiss 3 mm. Apochromatic Oc. 3.)

FIG. 2.

Periphery of right optic tract consisting chiefly of largish fibres. $\frac{1}{3}$ in. obj., Oc. 3.

FIG. 3.

Centre of right optic tract consisting chiefly of small fibres. $\frac{1}{3}$ in. obj., Oc. 3.

FIG. 4.

Section through the centre of chiasma (6 μ in thickness).

PLATE.

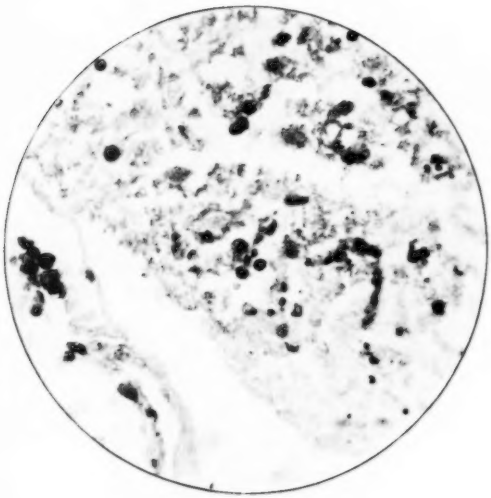

FIG. 1.

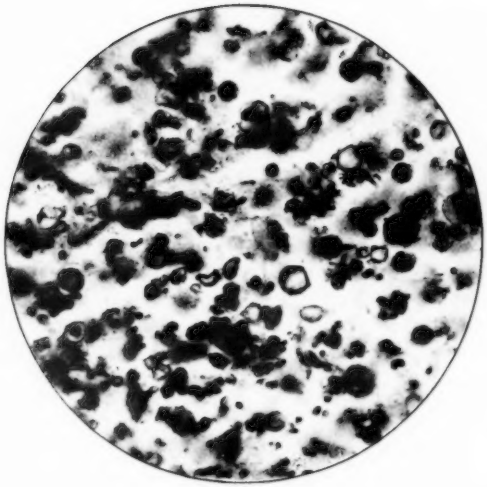

FIG. 2.

PLATE.

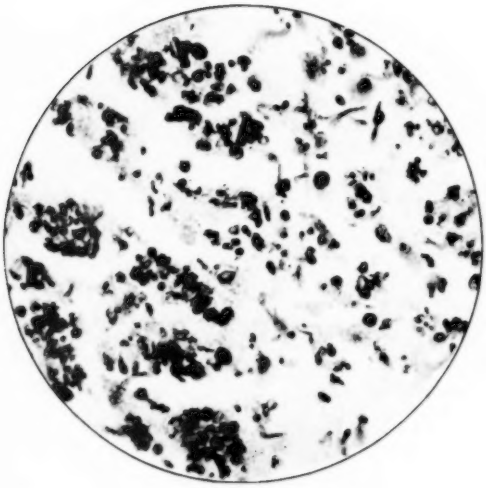

FIG. 3.

FIG. 4.

CONSIDERATIONS OPPOSED TO THE "NEURON THEORY."

BY ALEX HILL, M.A., M.D.

Master of Downing College, Cambridge.

THE literature of the neuron has been so frequently summed up during the last three years that it would be a waste of space for me to repeat here what is familiar to all neurologists. Dr. Barker's excellent treatise on the nervous system published at the beginning of this year, brings the history of the neuron controversy up to date.

It should be remembered, however, that the "Neuron Hypothesis," "Neuron Theory," or "Neuron Concept" is not, properly speaking, a theory at all. It explains nothing. It is a statement, supposed to be true, that every nerve-cell, with all the parts that belong thereto (axon, collaterals, terminal arborisations, dendrites) is anatomically distinct from every other nerve-cell. The conclusions with regard to physiological autonomy and the restriction of pathological processes which are often regarded as deductions from the "Neuron Theory," are really deductions from the Cell Theory of Schwann, and would equally hold good if all the neurons of the body were continuous one with another. When Hooke, in 1696, examining a slice of bottle cork, observed that it is divided into chambers like the "cells" of honeycomb, he supposed that the partition walls of these chambers were impervious. When, in 1839, Schwann applied the "cell theory" to animals, he imagined all plants and animals to be colonies of separate and distinct "cells." The discovery by Sachs, Gardiner and Russow that a plant is not, as Schwann supposed it to be, a colony of

separate cells, but a single protoplasmic unit imperfectly divided into cells by perforate cellulose septa, does not in any degree invalidate the cell theory. The discovery that the substance of any given cell is united by myriads of protoplasmic threads with the substance of the cells by which it is surrounded does not affect our conviction, based on innumerable observations, that every cell grows, lives and dies as an individual. The trophic individuality of the nerve-cell or "neuron" is a matter of observation. We have no reason to think that if every neuron were continuous with other neurons at a multitude of points the trophic control of one neuron would extend to any parts of its neighbours. Nerve-cells are uninuclear. Each nucleus is therefore the capital of a cell territory. Whether or not the economic processes carried out within the territory are in any way controlled from the capital, it is quite certain that each territory is self-governing. It may be easier to grasp, in the case of discrete cells, the fact that the metabolism of the territory stops at its boundary, than it is to realise that it stops at the histogenetic boundary of confluent cells; but we are acquainted with many tissues in which the cells are in various ways and in varying degrees united together, yet in none of these is there reason to think that confluence of structure indicates co-operation in nutritive processes. Each individual cell grows, carries on the business of its life, and dies, as a unit.

The belief that the neurons are discrete is based upon the appearances presented by chrome-silver and methylene-blue preparations. As to the distinctness of the coloured elements there can, as a rule, be no manner of doubt; but even here the rule is not absolute. I have found instances of the union by coarse bridges of two or even three cells in all classes of nerve-cells. Dogiel, Masius and others also maintain that, in certain cases, the dendrites anastomose. But such a union of homologous elements is of little interest. It merely points to their origin by cell-division. The neuron-statement is based upon the supposition that heterologous elements are never directly united together. Chrome-silver and methylene-blue preparations appear to

demonstrate the structural isolation of heterologous elements. Histologists have accepted their evidence as conclusive on this point. In a series of papers published since 1885, and especially in my presidential address to the Neurological Society of London, in 1896,[1] I have urged that the credibility of these witnesses should be further tested before their evidence is accepted as conclusive. At one time neurologists held, universally it may be said, a contrary opinion. Since the publication of Apáthy's paper in 1897 it has been recognised that, for invertebrates at any rate, the neuron-statement is untrue.

The union of homologous elements by means of their dendrites not being to the point, there are only two ways in which neurons can be mutually connected. (1) The axon of a central cell A may divide to pass without arborisation into several smaller (distributive) cells B; or, in other words, the apparent axons of several small cells B may be the subdivisions of the axon of a larger cell A: (2) The terminal arborisations of the axon of A may be continued into the dendrites of B. I shall endeavour to prove that unions of both kinds occur.

In recent years histologists have shown an ever increasing tendency to lay stress upon the direct connection of basket-like arborisations with the bodies of nerve-cells. Some histologists appear, indeed, to look upon the various forms of "basket," "pericellular network," and "pericellular tangle" as the chief means by which neurons are linked together. The subject is one of the greatest difficulty. But it appears to me on general grounds highly improbable that the basket is the most important terminal apparatus through which one neuron influences another. Phylogenetically it cannot be so: for it is through the neuropil that the branchings of associated neurons are first connected. Again, it is not a universal method of association, since no baskets are described as surrounding some of the most important cells, those of the spinal cord, for example. Looking at the matter from the opposite point of view the widely

[1] Hill. "The Chrome-silver Method," BRAIN, 1896, p. 1.

branching dendrites of such cells as the pyramids of the cortex or cells of Purkinje must have a significance, as collecting apparatus, which far surpasses that of any system of direct cell-body endings. The growth of a Purkinje cell is most instructive in this respect. When it is first recognisable as a distinct element (fig. 27) it is an oval cell from which an axon is already beginning to grow out. Soon its cell-body seems, if the chrome-silver method is to be trusted, to receive afferent filaments (fig. 28); at any rate it is always shaggy at this stage. Then with great rapidity its copious branching system of dendrites extends towards the surface —coarse and woolly as if connected with multitudes of fibrils—while the cell-body becomes clean and free from filaments (fig. 29). It seems to me incredible that any direct ending of nerve-branches in or around the body of a Purkinje cell, if there be such endings, can compare in importance, as a mechanism of association, with the elaborate dendrites of the cell.

Amongst descriptions of arborisation-endings within cells those of Held [1] are the most precise. Using iron hæmatoxylin staining Held exhibits certain minute rod-like particles or "neurosomes," which are closely aggregated in the axon and its terminal ramifications, and more widely scattered in the neuroplasm of the cell-bodies. By their aid he is able, as he believes, to follow the nerve-terminals given off by the fibre-baskets, which surround the cells of the nucleus trapezoides and other nuclei, into the substance of the cells and their dendrites. He shows these filaments entering the cell-bodies and penetrating even as far as their nuclei. But he believes that, having entered the cell-body, these filaments end abruptly, and do not pass out of it through its axis-cylinder. Held is therefore an upholder of the neuron theory, *i.e.*, of the theory of the discontinuity of the neurons, with this modification: that the end-branches of one neuron may actually penetrate into the body substance of others.

Had Held used a precipitable stain only, we should have discounted his results by the considerations—(1) that his

[1] Held. "Beiträge zur Structur der Nervenzellen und ihrer Fortsätze," *Arch. f. Anat. u. Physiol.*, Anat. Abth., 1897, p. 204.

fixing agents—alcohol, chloroform, acetic acid, and picro-sulphuric acid—cause the formation of coagulation products, which may have no existence in life, and yet be coloured after death—this is an objection to which Held himself calls attention; and (2) that iron hæmatoxylin gives a solid deposit in the cell substance. There is no problem in minute anatomy more puzzling than that of the weight to be assigned to precipitates within tissue elements as indicative of structural differentiation. For example: I have endeavoured to check Held's results by using Weigert's hæmatoxylin method after carmine-alum, and then over-bleaching the sections in ferrid-cyanide of potassium. Cells so treated are seen to be full of black granules, but the granules are not rod-shaped like the "neurosomes," nor can I detect any indications of a differential arrangement. Indeed, the size of the granules, which are always round or oval, and their condition of aggregation appear to depend upon the exact treatment to which they have been exposed.

Held's results have not been left unchallenged, as may be seen in a paper by Veratti,[1] which is illustrated with very striking figures of the author's own results. Turner and Hunter[2] also, using methylene blue, *intra vitam*, conclude that the filaments of the pericellular network do not enter the cell-bodies but lie upon them, and are continued on to their protoplasmic processes. The whole subject is one upon which it is permissible at present to retain an open mind. Especially is it legitimate to consider that the nature of these pericellular baskets is not yet definitely settled, when one reflects that neither Held's method nor the methylene blue exhibit the conducting fibrils of the nerves, but only the neuroplasm in which they lie. For my own part, I am inclined to believe that Turner and Hunter are right in supposing the fine nerve-filaments to lie upon the body substance of the cells which they invest. It appears to me that when nerve-fibres are split into their constituent

[1] Veratti. *Sul alcuni particolarità di struttura dei centri acustici nei mammiferi*, Pavia, 1900, pp. 81, 7 plates.

[2] Turner and Hunter. "On a Form of Nerve Termination in the Central Nervous System demonstrated by Methylene Blue," BRAIN, 1899, p. 123.

fibrils, or fine bunches of fibrils, the nutrition and insulation of these fine fibrils, which doubtless consist of conducting elements invested in neuroplasm, is in some way provided for by bringing them into juxtaposition with the cells near which they are passing, and the large dendrites of these cells. The neuroplasm of these cells is in some way favourable to the vitality of the fine fibres. It provides for the insulation and nutrition of the fibrils throughout the distance through which they lie upon it. To this subject I shall recur when speaking of granules.

With these preliminary observations I pass to the consideration of the question which I propose to discuss in this paper, namely: *Are the neurons genetically and structurally discrete; or do the distributing processes of one neuron form true anastomoses with the collecting processes of other neurons?*

As evidence of union of neurons I propose to consider:

(1) The relation of the terminal arborisation of axons to so-called "granules."

(2) The neurons of the sympathetic system.

(3) The evidence that "thorns" are portions of conducting fibrils surrounded by the neuroplasm of the collecting processes of nerve-cells.

(1) Granules.

When the chrome-silver method first came into use the extraordinarily distinct pictures of large nerve-cells which it presented in the cortex cerebri et cerebelli, the spinal cord, the olfactory bulb and elsewhere, attracted universal attention. They were so sharply defined and apparently so complete that they distracted attention from the other elements of which nerve-tissue is composed. Even now we frequently meet with an attempted counting of the "nerve-cells" in the cortex or in the central nervous system as a whole, in which no account is taken of the granules and other small cells! Yet the granules are far more numerous than the pyramids or cells of Purkinje, and each granule is equally complete, and equally entitled to the designation of

"nerve-cell" or "neuron." When Pedro Ramón showed the axons of the granules of the cerebellum, and his brother, S. Ramón y Cajal, the axons of the granules of the retina, they demonstrated that each of these granules is a perfect nerve-cell, with dendrites and axon and all other features of a neuron. They differ from the great cells which give off efferent fibres in no respects other than size. I have shown that the granules of the olfactory bulb (which other histologists set aside as "amacrine" cells or even as gleial cells), have axis-cylinder processes and thorny protoplasmic processes, and are therefore also complete nerve-cells;[1] a fact of some importance, apart from its particular application, because it leaves it open to us to conclude that when, in the future, we see a cell which has all the ordinary characters of a nerve-cell, and especially when it bears branching *thorny* dendrites, it cannot be a gleial cell, however difficult we may find it to demonstrate the axis-cylinder. As further evidence of the nervous nature of the granules of the olfactory bulb Blanes[2] has pointed out that they readily take the vital methylene-blue stain, whereas the gleial elements remain uncoloured; and further that they are to be found with their characteristic form in vertebrates which do not possess in this situation any typical gleial cells. It is clear that no plan of the structure of nerve-tissue is complete which does not include the granules, or which, in any situation in which they occur, sets them aside as non-nervous elements. Indeed they are just as important elements or units as the largest nerve-cells.

When a nerve-fibre reaches a muscle-fibre, or the modified muscle-fibre of an electric organ, its terminal arborisation, or the sarcogleia in which it is embedded, contains nuclei. In the end-plates of the frog these nuclei, which rest immediately upon the nerve-twigs, were termed by Henle the "nuclei of the branches of the axis-cylinder,"

[1] Hill. "Notes on Granules," BRAIN, 1897, p. 125. It was only after making thousands of preparations that I was able to demonstrate the axis-cylinders; and so curiously capricious is the chrome-silver colouration in this matter that, although I am always glad to show the sections in which axons are visible, I am afraid to part with them, since I have but two or three!

[2] Blanes. "Sobre Algunos puntos dudosos de la estructura del bulbo olfatorio," *Riv. trimestr. microgr.* 3, 1898.

and distinguished by him from the nuclei of the sheath of epilemma. No one, I think, will deny that there are, in the end-plate, nuclei which are different in appearance to those of the muscle substance. There are two, if not three, forms of nucleus on the fine nerve-twigs, which are distributed to the electric organ of the skate; but I do not find it possible, after allowing for the nuclei of the sheaths of the nerves (after they have lost their myelin), to distinguish with certainty any nuclei which clearly belong to the nerve-twigs. It seems to me quite possible, however, that Henle's nuclei of the branches of the axis-cylinder are nuclei of nervous elements through which the conducting fibrils of motor nerves are distributed to muscle-substance. If this conjecture be justifiable the conception of a motor neuron is incomplete unless the minute cells (daughter cells of the large cell in the anterior horn of the spinal cord?) are included in the neuron. This is equivalent to saying that the theory of the individuality of the neuron breaks down. I lay no stress upon this hypothesis of the mode of ending of motor nerves, however, but use it merely as an illustration of the mode of ending of ascending (sensory) fibres which I am about to describe.

The crucial test of the neuron theory lies, as it seems to me, in the proof that fibres which ascend from cells in the cerebro-spinal axis end freely in the cortical fields. If the ascending fibres pass without breach of continuity from cells in the spinal ganglia or in the nuclei of Goll and Burdach into cells in the cortical field—if, that is to say, they start in one large cell (or, accepting Apáthy's findings, if they originate with a "fibre-cell," pass through a "ganglion-cell") and join several small cells—the neuron theory falls to the ground. It is true that various qualifications are introduced by recent writers into the expression of the theory; "relative individuality of the neurons" is often substituted for the definite expression of absolute independence; but if as a mechanism each nerve-cell with its collecting and distributing processes is not an anatomical integer but a part of a chain of cells, the theory loses all significance.

It is generally assumed, in the case of the cortex cere-

belli at any rate, that afferent fibres come into relation with "granules" and that the axons of the granules transmit the impulses, which the afferent fibres bring, to the more superficial elements of the cortex. Ramón y Cajal represents the branching afferent fibres (mossy fibres) as being in relation with the clawed dendrites of the granules, and the bifurcating axons of the granules as in relation with the dendrites of the Purkinje cells. Neglecting all other elements and reducing the scheme of connections to a diagram, the conducting arc of the cortex cerebelli consists of a mossy fibre—its end-branches, granules; their axons, dendrites of a Purkinje cell—its axon. The granules are, according to this scheme, secondary or tertiary neurons of the chain leading from sensory cells to the cortex cerebelli, and each granule is supposed to be as independent, as self-contained, as the larger neurons between which they are intermediaries.

In my preparations I find a few instances of granules which (figs. 1, 2 and 3) are distinctly connected with fibres of the arbor vitæ on the one side and with divaricating axons, passing between the cells of Purkinje to the molecular layer, on the other side. I find a very large number of granules which are connected with fibres from the arbor vitæ. In some cases the axon of the granule passes with a much twisted course towards the arbor vitæ (figs. 4, 5). In the two specimens figured (4, 5) the connection of the granule and the nerve-fibre occurs at a point which will afterwards become the clawed end of one of the arms of the granule.[1] Fig. 6 shows a granule the axon of which joins, apparently, a branch of a fibre from the arbor vitæ which ends by ramifying in the molecular layer. Figs. 7 and 8 illustrate a not uncommon disposition of the axon of a granule in which, before it reaches the sheet of Purkinje cells it bifurcates, giving one branch to the molecular layer and another which runs inwards towards the arbor vitæ. In fig. 9 is seen a granule the axon of which has an extra-

[1] It will be understood by everyone who has worked with the chrome-silver method that when I assert that a fibre is continuous, or that two fibres are in connection, I wish to imply that all attempts to prove discontinuity by careful focussing, by oblique illumination, &c., have failed. When two opaque black objects are superposed or in close juxtaposition, they appear to be in continuity. It is never possible to make sure that this is the case.

ordinarily tortuous course and gives off several branches in the granular layer as well as one or more branches to the molecular layer.

It will be noticed that none of the granules figured have the ordinary form of the cerebellar granule. This, as is well known, is a round cell with four or five dendrites ending in clawed hands and a single axon which divaricates into two long fibres in the molecular layer. But granules of this form do not make their appearance until comparatively late in development; in puppies at about 18 days, kittens at about 14 days, hedgehogs at about 18 days. It is, I fear, impossible to give exact dates owing (1) to the unreliability of the chrome-silver method, and (2) to the fact that the histogenetic development of the cortex cerebelli takes place gradually. Granules having the immature, carrot-like form represented in figs. 1 to 8 are to be found long after the first mature granules have made their appearance. To make this part of the subject clear it will be necessary to give some account of—

The Development of Ordinary Granules.

In a paper published in 1889 Bellonci and Stefani[1] gave an accurate account of the changes in the external nuclear layer, which might have suggested the migration of the granules subsequently discovered by Rámon y Cajal. They described the formation of a molecular layer containing some scattered cells before the overgrowth occurs, from behind forwards, of the nuclei of the ependymal stria of the velum medullare posterius. As the nuclei spread forwards from this stria (the Rautenlippe of His?) their mitotic figures indicate active division. For some time before the appearance of the cells of Purkinje, and still more after these are differentiated, the nuclei are distinguishable into two zones—an external, in which the nuclei are round and rich in caryokinetic figures; and an internal, in which the nuclei are elongated transversely and rarely show caryo-

[1] Bellonci et Stefani. "Contribution à l'histogénèse de l'écorce cérébellaire," *Arch. ital. de Biologie*, xi., 21.

kinetic figures. The molecular substance is chiefly made up of transverse fibrils which Bellonci and Stefani presume to be derived from the elongated cells. "The deepest nuclei of this layer appear uncoloured and are consequently seen with difficulty. As the molecular layer grows in thickness they are destroyed in great numbers." Löwe[1] had previously described the molecular substance as formed by the fusion into a mass of the cell bodies of these nuclei, the nuclei becoming vesicular and gradually disappearing.

In the light of Rámon y Cajal's observations, to which reference will be made immediately, it is clear that the disappearance of the nuclei is due, not to their degeneration, but to their migration past the cells of Purkinje into the granular layer.

Simultaneously with Bellonci and Stefani, Lahousse[2] described in very similar terms the appearance and overgrowth of the external nuclei. He named this layer "the layer of Obersteiner," since the Viennese professor was the first to describe it;[3] but he prefers to call the cells of which it is composed "cellules conquérantes" or "cellules de renfort." "The reinforcing cells multiply abundantly and invade, from behind forwards and from within outwards, almost the totality of the external surface of the cerebellum, with the exception of the median line and the posterior part of the inferior vermis, in the neighbourhood of the median line."

It is evident from the descriptions just quoted that the cells of the external granular layer have a secondary origin. They are not formed from the general mantle-layer of the cerebellum. It is curious and probably significant that their site of origin should correspond nearly with that of the spinal ganglia, and that each little cell should be in the mode of its growth—a fusiform cell converted into a round cell with an axis-cylinder in the form of a T—a root-ganglion

[1] Löwe. *Anat. u. Entwicklungsgeschichte d. Nervensystems*, vol. ii., Leipsic, 1883.

[2] Lahousse. "Recherches sur l'ontogénèse du Cervelet," *Arch. de Biologie*, viii. (1888), p. 43.

[3] Obersteiner. "Beiträge z. K. von feineren Bau der Kleinhirnrinde," *Sitz. d. K. Akad. d. Wissensch*, Vienna, 1860, and again in *Biologisches Centralblatt*, iii. (1883).

cell in miniature, *plus* five or six clawed arms. We owe to a very remarkable piece of histological work carried out by Rámon y Cajal the proof that the granules undergo the transformation just described.

According to Ramón y Cajal, the precursors of the granules are fusiform cells, tapering into a fibre at each end (figs. 18 and 19).

That Ramón y Cajal's description of the transformation of fusiform cells into granules is right is borne out by appearances such as are shown in figs. 18, 20, and 22, as well as by the fact that the fusiform cells are not to be found in adult cerebella. There are, however, some difficulties in explaining how the one kind of cell is transformed into the other.

It is, for example, to be noticed that in the embryonic state of these granules—when they are fusiform or "fibre-cells"—there is an obvious distinction between the thread-like axon on the one side and the thicker process on the other side, which has the appearance of a dendrite (fig. 18), and yet both alike, according to Ramón y Cajal, become horizontal limbs of the T-shaped axon of the granule. Further, certain noteworthy physiological difficulties result from this. In the first place, since the granules cannot be supposed to have any function until they have, by sinking beneath the layer of Purkinje cells, established connections with afferent (mossy) fibres, it is remarkable that their processes should be so long. The length of the tangential fibres, to which the fusiform cells which Ramón y Cajal regards as embryo granules give rise, is very remarkable. I have traced them in a puppy for fully $1\frac{1}{2}$ mm. A study of the histogeny of the nervous system leads to the conclusion that the growth of conducting strands, if it cannot be described as a response to function, keeps but little, if at all, ahead of function. Yet here we have what, to all appearance, are well-developed nerve-cells and fibres which cannot, so far as we can understand the conditions which obtain in the embryonic cerebellum, take on functional activity for some time. We should have expected to see the lateral extension of the limbs of the T delayed until there was a

possibility of impulses passing into them up the vertical limb. In the second place, if the view universally held as to the function of the granules—that they collect impulses from the afferent (mossy) fibres and distribute them to the dendrites of the Purkinje cells by means of their divaricating axons, a view enunciated by Ramón y Cajal in his Croonian lecture—be correct, the distinction between cellulipetal and cellulifugal conduction, in the case of granules, breaks down. Both the axon and the dendrite of the precursor of the granule conduct (when they become the two limbs of the T-formed axon) outward from the cell. It may be said that a similar difficulty has to be met in the case of the cells of spinal ganglia; but the cases are not quite parallel. It is open to us to consider the distal process of a root-ganglion cell as the dendrite, the proximal process as its axon; since the one conducts cellulipetally, the other cellulifugally. Of course, if Apáthy's theory of "nerve-cells" and "ganglion-cells" be substantiated, this point loses its significance, since the root-ganglion cells must be "nerve-cells," from which fibrils are developed, which grow both outwards to the sensory cells and inwards into the axial cells. If the description given by Ramón y Cajal of the formation of the granule be correct, the cell-body withdraws from the fibre, just as in the case of a root-ganglion cell; but the horizontal limb of the T is not a direct conductor from left to right, but a distributor of impulses to the left and to the right.

Here I ought to mention a difficulty which has met me in tracing the origin of granules from these fusiform cells. In long hardened tissue I find these fusiform cells—which might be termed "fibre-cells"—in the developing molecular layer in very great numbers; but I have never succeeded in demonstrating the first step in their conversion into granules. I find granules with a carrot-like apical process which points towards the granular layer, and a very short T-process which branches right and left in fine nerves (fig. 20), which looks as if it might have been derived from a fibre-cell; but after a most prolonged search, I have failed to see the first step in the pinching together of the two processes of a fibre-cell. Again, in certain animals, especially

the puppy (fig. 18), the fibre-cells appear to be larger and better developed than the first-formed granules. Lastly, these well-developed fibre-cells often show fine branches given off vertically to their lateral continuations, both axonal and dendritic (fig. 19). Such branches appear to have no place in the constitution of the granules.

Athias[1] insists that the bipolar cells are at first connected with the surface by a thick vertical process. Ramón y Cajal figures the same condition. But as soon as the body of the granule has withdrawn from the horizontal fibre it shows a thick process directed towards the granule layer. This reversal is certainly very remarkable, and I have looked carefully for transitional stages, but without success.

Occasionally I find a developing granule with a thick apical process which points towards the surface; but this condition is rare, and I think that Cajal, Popoff, Athias, and others, have confused with developing granules cells of a different kind (figs. 15, 16, 17) which are to be found in the deeper part of Obersteiner's layer, or just below it. Under any circumstances it is very difficult to understand the transitions through which a truly bipolar cell (fig. 18) passes in becoming a granule. At first it has an axon to one side and an apical process to the other. Then, according to the received account, it has an apical process directed towards the surface, and axons to right and left. Next it has a centrally-directed apical process and axons right and left. It appears to me that three different kinds of cell are developed in the molecular layer or superficial to it—A and B, the granules which I have called carrot-granules, and the ordinary granules from Obersteiner's layer—C, the cells of Cajal, either from the deepest part of Obersteiner's layer or from the underlying superficial part of the mantle-layer.

Our information with regard to the histogenesis of the cerebellum is far from complete, but the following points seem to stand out clearly: (1) The nuclei found at birth and for some time subsequently, outside the sheet of cells of Purkinje in the cortex cerebelli, are much more numerous

[1] Athias. "Recherches sur l'histogénèse de l'écorce du cervelet." *J. de l'Anat. et de Physiol.*, XXXIII. (1897), p. 372.

than the nuclei of the molecular layer in the adult. (2) There is no sufficient indication of cell division on the ventricular side of the cells of Purkinje to account for the formation of the granular layer. Hence the granules are migrants from the superficial tissue. (3) From Obersteiner's layer are developed granules, "carrot-granules," Cajal's cells, and the small cortical cells or stellate cells of the molecular layer. (4) In the mantle proper are developed gleial cells, Golgi cells, and Purkinje cells.

The further stages in the growth of the granules have not been described, but they are of great interest. As soon as the granule has passed through the layer of Purkinje cells its apical carrot-shaped process increases considerably in length and gives off several slender side branches (figs. 1, 3, 23) which divide two or three times into long slender twigs. In this stage it shows none of the clawed arms which are so characteristic of the fully formed granule. These appear later as lateral branches, very short at first (fig. 25). In the meantime the long apical process makes its way towards the arbor vitæ, which it frequently enters, becoming lost among the fibres (fig. 1). Its appearance at this stage is strongly suggestive of its feeling down amongst the fibres for nerve connections.

In puppies until they are about 18 days old, hedgehogs 18 days, kittens 2 weeks, none of the typical round granules with four or five arms ending in claws are to be found. All the granules have the appearance shown in figs. 1, 2, 3, &c. It is at this stage that the granules show a double connection—with the fibres of the arbor vitæ on the one side, and with an axon divaricating in the molecular layer on the other.

It is also to be noticed that the granules, which at first are placed with their long axes horizontal (fibre-cells), are next placed vertically, as they migrate past the cells of Purkinje, and lastly, more or less horizontally again as they approach the arbor vitæ.

The meaning of the "claw" is at present obscure. The claw assumes, in chrome-silver preparations, many different forms. Sometimes it appears as a hand with four or five fingers. Occasionally it is a complete basket, which is often

larger than the granule which it appears to enclose; too large, that is to say, to be in contact with the body substance of the granule which it envelops. Every intermediate condition between these two forms is seen. Every now and again one sees a basket which appears to re-form into a fibre, as if the arm of the granule which had divided into a basketwork were in continuity through this basketwork with a (? mossy) fibre.

In my paper on the Chrome-silver Method I called attention to the fact that the baskets of Ramón y Cajal which invest the cells of Purkinje are frequently seen to reunite beneath the cells into bunches of fibres, which enter the granular layer. In other words, the tassel of fibrils depends beyond the base of the Purkinje cell. Instead of the tassel of fibres being closely applied to the surface of a Purkinje cell it is loose and large, and its twigs surround the axon of the cell. This observation is confirmed by Dr. John Turner, staining with methylene blue.[1] On the other hand, fibres which branch in the granular layer break up into baskets which approach the cell from below. Fibres are frequently seen in the granular layer which have just the same relation to the Purkinje cells as the branches from the axons of Cajal's cells, except that whereas the latter approach them from below, the former approach them from above. It is quite possible that the two kinds of basket are continuous, or, to speak more correctly, that there is only one kind of basket into which the fibre from the one side breaks up, and from which the fibre on the other side is re-formed; the chrome-silver method revealing sometimes the one half of the basket, and at other times the other. It is very difficult to account for such an arrangement as a basket-work which envelops the body of a cell without establishing any connections with it; and I hesitate to suggest a hypothesis, but it seems to me not impossible that where connection is to be established between two kinds of nerve-fibres the fibres break up into their constituent fibrils, which rest upon the body of a neutral cell. If this be possible, the

[1] Turner. "A Note on the Staining of Brain in a Mixture of Methylene Blue and Peroxide of Hydrogen." BRAIN, 1900, p. 524.

cell of Purkinje in some way provides for the insulation and nutrition of the isolated fibrils. In the same way the baskets into which the arms of the granules appear to end may be bunches of elementary fibrils separated out at the spot where the arm of a granule joins some other kind of fibre. I shall amplify this hypothesis later on.

Carrot-Granules with Centripetal Axis-Cylinders.

In 1896[1] I described and figured granules whose axis-cylinder processes join the fibres of the arbor vitæ. They are distinguished by their carrot-like form, and are only found in tissue which has been hardened in bichromate (with or without osmic acid or formalin) for a long time.[2] Subse-

[1] BRAIN, 1896, p. 35, fig. 14.

[2] *Method.*—All the sections figured in this paper were prepared from tissue which had been immersed in bichromate of potassium and osmic acid or formalin for a long time. When endeavouring to ascertain the nature of the chrome-silver reaction I varied the length of time during which the tissue was immersed in the respective fluids within very wide limits. This research was undertaken without any thought of discovering new elements of nerve-tissue, but merely with a view to ascertaining the nature and the limitations of the method. Unlike Lenhossék, Ramón y Cajal, and others, I find that nothing is lost by a long immersion in the chromate. I know of no structural feature which is exhibited in tissue hardened in bichromate of potassium and osmic acid for a few days, which is not also to be found in tissue which has been hardened in this mixture for two or three years before being transferred to silver nitrate. On the other hand, I have never found "carrot-granules" or the most delicately-branched fibres of the granular layer in tissue which has not been hardened for at least a month; and I am disposed to think that they are best seen in tissue which has been hardened for at least three months.

After a year the tissue becomes very brittle and difficult to handle, and since nothing more is to be gained by such a prolonged hardening, I regard the best length of time for immersion in the chromate as from three to six months. This method is especially successful with young animals—cats, dogs, &c., of not more than a month old. Among my specimens from adult brains which have lain in bichromate of potassium and osmic acid for more than a year the greater number are complete failures. Some, on the other hand, are admirably coloured. Evidently, therefore, other conditions beside the long hardening help to determine the success or failure of the impregnation. How admirably it may succeed is well shown in fig. 12, of a Golgi cell from the cerebellum of a cat twenty-seven days old, hardened for five months. This cell lies at the apex of a convolution, and it is curious to note that its branched axis-cylinder is continued by a very delicate fibre to the extreme base of the convolution, a distance of at least 2 mm. Whether or not this revelation is due to the method it is impossible to say, but I have never seen such a connection in a specimen hardened as directed by Golgi or by Ramón y Cajal. Nor is it in accordance with any description of these cells of Golgi's type II., the specific character of which is supposed to lie in the complete arborisation of their axons within the cortex.

It is to be noticed that the dendrites of the Golgi cells are destitute of thorns. In this respect they contrast in a very marked way with the extremely thorny dendrites of the Purkinje cells, fragments of which are to be seen on the right and left of the figure.

quently (BRAIN, 1897, pp. 125 and 466) I described and photographed granules of extremely similar form, which I had found in the molecular layer. After Ramón y Cajal's description of the origin of the granules from bipolar cells, I am satisfied that those which I described are embryonic granules, strangely unlike to adult granules though they are. Carrot-granules with centripetal axis-cylinders are, however, cells which cannot be confused with developing clawed granules. They are most numerous at the apices of the folia, when they appear (as shown in fig. 13) as cells of considerable length. They always take a brown colouration, the nucleus remaining clear. Their apical processes divide two or three times as they extend outwards towards the cells of Purkinje. Their axis-cylinders may be undivided, or they may give off branches which return towards the surface. In the developing cortex cerebelli, at an early stage, cells are to be seen which cannot be embryonic granules of the ordinary form (figs. 15, 16, and 17). They are placed vertically to the surface. Their apical processes reach at first to the pia mater, where they usually end in a knob, which rests against this membrane. Their axons take a centripetal course. Subsequently the apical process withdraws from the pia mater, and the cells sink more deeply in the molecular layer. Their axons frequently give slender lateral branches. It is possible that these cells will grow into Golgi cells or even into cells of Purkinje; but considering the very early date at which cells of both these classes assume a well-developed appearance, I am disposed to think that the fusiform cells just described are not destined to undergo this transformation, but are developing carrot-granules. The cells represented in fig. 17 in the molecular layer certainly appear to be the same as those which are represented in fig. 13 in the granular layer.

I have found carrot-granules in very large numbers in rats (up to 12 days), kittens (to 14 days), puppies (to 21 days), hedgehogs (to about 18 days). I have never found them, with certainty, in adult cerebella. It is true that I have found a few doubtful specimens in adult sheep, but they were not sufficiently distinct for me to lay any stress

upon them as proving that the granule with centripetal axis-cylinder is a permanent structure. On the other hand it should be noted that these granules make their appearance only after long hardening, and I have but very little adult tissue which has been so treated. One is afraid to lay down any rule for the chrome-silver reaction, the next set of sections prepared may prove it fallacious; but in several specimens of tissue which were tested by the nitrate of silver at intervals during the hardening process, the granules with centripetal axons were not found until after long hardening, the earliest case being after immersion for five months in bichromate and osmic acid.

Are the carrot-granules with centripetal axons, like the carrot-granules with centrifugal axons, embryonic cells? A very large number of observations have failed to reveal any transitional forms, and it is difficult to conceive, if they be embryonic, into what adult elements they grow. It might be supposed that they grow into Golgi cells; but the Golgi cells are remarkably mature in appearance in sections which abound in carrot-granules; indeed, they are amongst the earliest of the cerebellar elements to take on an adult form. Again, the Golgi cells are always supposed to belong to "Golgi's type II.," that is to say they are cells with short axis-cylinders. (A curious instance of an axis-cylinder which does not terminate by arborisation in the vicinity of the cell is shown in fig. 13; but this does not justify us in drawing the conclusion that all Golgi cells have connections with the fibres of the arbor vitæ.) I cannot at the present time see any indication that the carrot-granules are young Golgi cells. The only alternative left open is, therefore, that either they are cells found in the young animal which disappear in the adult, or that they are cells which the chrome-silver method reveals in the young animal (owing to its peculiar suitability for embryonic tissue) and does not reveal in the adult.

If these cells belong to a distinct and hitherto unrecognised class it is most important that they should receive a distinctive name. The application to tissue elements of the names of their discoverers is to be deprecated except in very

special cases. That Purkinje, Golgi and Cajal should have written their names on the cerebellum is no more than their due, but even in their case the application of the name should be rigidly limited. The cells most intimately associated with Cajal are the basket cells. Cells of the molecular layer which do not form baskets must bear another name, and probably none is better than "small cortical cells," proposed by Schäfer. Golgi's cells are the remarkable large cells with branching axis-cylinders. Some of them lie amongst the cells of Purkinje and branch in the molecular layer, whereas others are restricted to the granular layer. The application of Golgi's name to other cells, and especially to the granules, is extremely confusing. Probably the time-honoured name "granule" is the least likely to be misunderstood. If the cells which I have described are distinct from the typical round granule, the term "carrot-granule" will probably as well as any other mark this distinction and at the same time convey to the reader's mind the idea that they are so small as to belong to quite a different class of element from the Golgi-cells. Their rarely-branching axis-cylinder continued into the arbor vitæ completely separates them from the "cells with short axis-cylinder of Golgi's type II."

Since the cells of Purkinje are the only recognised cells with descending axons, I have spoken of the carrot-granules as connected with ascending fibres; but this is, of course, a hypothesis in support of which we have not, at present, any evidence.

(2) Sympathetic System.

When evidence derived from the central nervous system led to the formulation of the neuron theory, it was concluded that the cells of the sporadic ganglia must be brought into line with the neurons of the axis. Instead of the sympathetic cells being "distributive," in the sense that each receives one nerve-fibre of a certain thickness and gives origin to a number of thinner fibres—the afferent fibre being usually medullated and the efferent fibre non-medullated—it was assumed that the thicker fibre is the

axon, the thinner fibres dendrites. But the histological evidence upon which this view is based is not by any means satisfactory. I may summarise my doubts as to its justice under the following heads: —

(*a*) The alleged dendrites do not present the characters by which dendrites are usually distinguished. This is shown in all figures with which I am acquainted (see, for example, Köllicker's "Gewebelehre," vol. ii., figs. 834, 835), and therefore I have not thought it necessary to prepare fresh illustrations. The end branches of the so-called dendrites are exceedingly slender filaments, often of a great length. They are usually varicose—a very characteristic feature of the finest nerve-fibres and a rare feature in dendrites. They are destitute of thorns. For reasons which will appear later I regard thorns as the distinctive markings of true dendrites, but on this point I possibly take too strong a position.

(*b*) The so-called dendrites may often be followed beyond the limits of the ganglion. Where stained cells lie near the point of exit of a gray ramus the so-called dendrites frequently pass as a bundle of very slender fibres into the ramus.

(*c*) If it be accepted that the branching processes are dendrites it is very difficult to assign to them any function. Setting aside the doctrine of their purely nutritive function, dendrites are regarded as establishing connections between the cells to which they belong and the end-branches of axons of cells lower or higher in the system. The tissue of which the ganglia are composed is however peculiarly unfavourable for such connections. It is a dense connective tissue packed with large nerve-cells and traversed by innumerable nerve-fibres. There is no "gray matter." There is none of the tissue which, in the cerebro-spinal axis or in the ganglia of invertebrates, seems to be essential for the establishment of such connections.

(*d*) Failing communications between end-branches and dendrites, the manner in which the neurons of the axis influence the neurons of the ganglia is insufficiently explained. It appears to be limited to the various forms of "basket" which have been described. Ramón y Cajal, Retzius, Kölliker and others, have described certain of the "dendritic

branches" as forming baskets, upon which Kölliker very properly remarks: "Welchem Verhalten ich jedoch bei genauer Ueberlegung keinerlei physiologische Bedeutung zuschreiben kann und dasselbe nur für etwas zufälliges erklären muss" (*loc. cit.*, p. 853). The whole question of baskets, pericellular plexuses, and pericellular tangles is most obscure. Their primary purpose does not seem to me to be to establish a connection between end-branches and the cell-body which they invest; to this question I have already referred, suggesting a hypothesis, when treating of the granules of the cerebellum.

(*e*) It is beyond question that the præganglionic nerves (white rami, &c.) contain fewer fibres, and these larger (medullated) than the postganglionic nerve. As Gaskell's[1] careful analysis of the sympathetic system showed, the ganglia may be looked upon as stations in which medullated fibres are replaced by bundles of non-medullated fibres. If the old view that the cells of the sympathetic ganglia receive each a large fibre and give off a number of fine fibres be discarded, the enormous multiplication of fibres which occurs in the ganglia must be effected by the subdivision of the axons of their cells. Such a subdivision is assumed; but in cases in which that process of a cell which is, beyond doubt, its axon can be followed for some distance, it is, so far as my own observations go, conspicuous for the absence of collaterals or side branches of any kind.

(*f*) If the neuron theory be applicable to the sympathetic system, which is, so far as we know, entirely an efferent system, the præganglionic fibres must end by arborisation, the axons of the cells must constitute postganglionic fibres. I have made various preparations with a view to ascertaining if this is the case. Owing to technical difficulties these preparations are not quite satisfactory, but they leave me in little doubt but that the axons of the cells of the sympathetic ganglia are directed towards the præganglionic fibres, *i.e.*, towards the cerebro-spinal axis. In the superior cervical ganglion, for example, the axons leave the ganglion at its

[1] Gaskell. "Nerves which Innervate the Visceral and Vascular Systems." *Journal of Physiology*, vii., 1.

cervical end. This is a question upon which I hope to give a further report in a short time.

(*g*) The mode of origin of the sympathetic ganglia is suggestive of a permanent connection between their cells and cells in the cerebro-spinal axis. The sympathetic neuroblasts are cells which at a very early stage have migrated from the neuro-epithelium which forms the cerebro-spinal axis and the spinal ganglia. It may well be that they retain, during their migration outwards, a connection with the cells with which they are genetically associated. They may each "pay out" a strand—the subsequent axis-cylinder which keeps them in union with their mother cell. This is, of course, a mere speculation. If the sympathetic rudiments are separated off from the rudiments of the spinal ganglia as His teaches, the genetic connection would be difficult to explain, since the fibres of the white rami communicantes travel chiefly in anterior roots. At any rate the fact of migration makes the retention of connecting fibres between mother cells and daughter cells possible, whereas, if the ganglia had completely independent rudiments, it would be impossible.

The strongest argument for discontinuity of neurons in the sympathetic ganglia is to be derived from the well-known observations of Langley and Anderson, that nicotin paralyses the ganglia, preventing the passage of impulses *viâ* the ganglion-cells, while it does not paralyse the fibres which pass without interruption through the ganglia. It would seem natural to suppose that the nicotin prevents the end-branches of the higher neuron from acting upon the cell-body of the lower neuron. But, on the other hand, against this interpretation is the fact that nicotin does not thus stop the passage of impulses in the ganglia of Auerbach and Meissner in the intestine.

Langley, on this account, doubts whether these are ganglion cells in the proper sense of the term. They have, however, exactly the same histological characters as the cells of the ganglia. This is shown in figs. 841, 842, 843, 844, 845, of Kölliker's handbook. A study of these intestinal plexuses reveals their amazing richness in nerve-cells. If these cells are stations for breaking up nerve-fibres

afferent to the plexus into bunches of efferent nerve-fibres for the supply of the musculature of the intestinal wall, it is not impossible that they provide a separate fibril for every muscle-cell.

If the neuron theory is true it is applicable to the sympathetic system. The evidence that this is the case is not, as yet, complete or convincing. There are, on the contrary, reasons for supposing that fibres which start from cells in the cerebro-spinal axis end in cells in the sympathetic ganglia, and that fibres from cells in these ganglia end in cells of the intestinal plexuses. If such a direct connection obtains between cell and cell, the neuron theory of the separateness and isolation of all nerve-cells breaks down.

(3) Thorns.

In papers already referred to I have called attention to the following facts :—(*a*) By varying the method of preparation, although using absolutely fresh and healthy tissue, specimens of cortical pyramids and other large cells may be obtained which in some cases are absolutely thornless, whereas in other preparations they bear thorns. (*b*) Thorns present a great variety of form. Usually they are short rods ending in knobs ; occasionally they appear as filaments without knobs, two or three times as long as the knobbed rods. (*c*) In certain specimens no thorns are seen, but their place is taken by one or more minute black dots. When there are several of these dots they are always arranged in a straight line.

From these facts I draw the conclusion that the substance which has the power of fixing the chrome-salt, and, therefore, on the entrance into the tissue of silver-nitrate, of forming a silver-chrome salt which is subsequently reduced,[1] is a semi-fluid substance which surrounds and in-

[1] The nature of the reaction which occurs in the Golgi method of colouration does not seem to be generally understood. The reaction appears to be as follows :—When nerve-tissue is placed in a chromate, certain cells and fibres fix the chrome-salt. When the block is dropped into nitrate of silver, the loose chrome-salt quickly diffuses out into the silver-nitrate, which it precipitates as silver-chromate. The silver-nitrate enters the block more slowly, and wherever it finds a tissue-element which has retained the chrome-salt it combines with the chrome-salt to form an unstable subchromate of silver. Osmic acid and formalin favour the fixation of the tissue, without in any way taking part in, or interfering with, the reaction.

vests unstaining fibrils. The usual appearance of a rod and knob is due to the fact that at the time of fixation the semi-fluid cytoplasm of the dendrites is extruded outwards along the unstained filament which every thorn contains. Beyond a certain distance from the dendrite the film, which presumably becomes thinner and thinner the farther it is from the dendrite, collects, under the force of cohesion, into a droplet. The thorn, therefore, appears as a rod ending in a knob, because this is the form which the stainable cytoplasm assumes; but there is no ground for supposing that the supporting unstained fibril ends at the knob. If a thin wire be plunged into syrup, the viscid liquid which forms a film around the wire at the moment when it is withdrawn, runs the next moment into drops. When a thorn is replaced by a row of black dots, it seems clear that the droplets are supported upon a something which resembles the wire which supports the drops of syrup.

Thorns, therefore, are not artifacts in any proper sense of the word. They are the unstained (conducting?) fibrils surrounded for a longer or shorter distance with the cytoplasm of the dendrites, the cytoplasm alone being coloured.

This hypothesis is justified by a study of the development of thorns. In young cells, in which the cytoplasm is, relatively to the nerve-fibrils, apparently more abundant than it is in adult cells, the thorns are larger and coarser. This is conspicuously the case on the woolly-looking dendrites of the cells of Purkinje at a very early stage. It may also be noticed, as was pointed out by Athias, that when the cells of Purkinje are first recognisable, filaments appear to join their cell-bodies; whereas, *pari passu* with the growth of the dendrites, the cell-body becomes clean and free from filaments, as if the dendrites carried the connecting filaments away from the body of the cell. Figs. 27, 28, and 29 represent three very typical stages in the development of a Purkinje cell.

If this evidence be admitted, gray matter contains a scaffolding formed of an infinite number of fibrils, which place the arborisations of axons and of collaterals in connection with dendrites. Owing to the extreme tenuity of

these fibrils in higher animals, no method of staining them outside the cell, in such a way that they are amenable to examination by the microscope, has yet been devised; although, as will be presently explained, Bethe seems to have stained either certain of the larger fibrils or groups of fibrils. Yet the delicate fibrils of vertebrate nerve-tissue are probably the phylogenetic successors of the coarser fibrils, which Apáthy, using chloride of gold, demonstrates in invertebrates. For the protection, nourishment, and insulation of such fibrils some "intercellular substance"—in the sense in which Nissl[1] uses the term—seems to be needed. Within the cell the fibrils are supported by cytoplasm.

If it be urged that the chrome-silver staining ought to reveal the presence of this labyrinth of primitive fibrils, it may be answered that it very frequently does do so; but all preparations in which there is evidence of the existence of an infinity of fibrils are set aside as mis-stainings. Everyone who uses the Golgi method is familiar with the blotches of black deposit, looking like a burr on a wild rose-bush, which are seen here and there in most preparations. They are set aside as "deposits" because no details of structure can be seen within them; but the fibrils which appear around the edge of the blotch are no doubt actual structures about which the deposit has occurred.

Further, it must be remembered that intracellular fibrils have been described by many observers. It can hardly be doubted that such primitive fibrils exist; and if—as there seems every reason for thinking—they are the conducting elements of the nervous system, it is much more likely that they come to the cell from outside than that they originate within the cell and its dendrites.

Since Max Schultze, in "Stricker's Handbook of Histology" (1871), described the fibrillation of the cell-body, fibrillæ have been described by H. Schultze, Flemming, Benda, Dogiel, Kronthal, Apáthy, Nissl, Held, Bethe, *et al.* No two of these descriptions exactly agree, so far as regards the size and disposition of the fibrils within the cell, and it

[1] Nissl. "Nervenzellen und graue Substanz," *Münch. med. Wochenschrift*, 1898, pp. 988, 1023, 1060.

must be admitted that it is impossible at the present moment to form a clear idea as to their nature. Bethe's[1] descriptions are the most definite. He stains the primitive fibrils with toluidin blue after solution of Nissl's bodies in ammonia. The fibrils are so closely set in the axons that it is difficult to distinguish them individually, although the fibrils destined for collaterals are recognisable owing to their greater distinctness and dark colouring. But by no means all the fibrils which join dendrites are destined for the axon. Some traverse the cell body on their road from one dendrite to another. Others again, enter by one branch of a dendrite and leave by another. Bethe's description differs from Apáthy's in one very important respect. He sees the primitive fibrils as distinct and separate fibrils, traversing the cell-body without anastomosis, whereas Apáthy finds that in the majority of cases the entering fibrils form a plexus in the periphery of the cell from which radial branches pass to the neighbourhood of the nucleus where they form a second plexus, out of which arises the, usually single, neuro-fibril of the axon. Neither Apáthy's nor Bethe's description fits in with the description of Golgi[2] of a comparatively coarse plexus in the periphery of the cell. This latter is not perhaps a very important point. From many preparations of Golgi's intracellular network which I have made, I am inclined to regard it, since it is sometimes coarser, sometimes finer, as not the demonstration of definite structural elements but a somewhat irregular deposition of silver in the "unstainable" substance of Nissl. It seems to me that the protoplasm in which the tigroids are embedded fixes the chrome-salt, and this subsequently combines with the silver, without regard to any primitive fibrils which may pass through it, except in so far as the arrangement of the fibrils determines the arrangement of the silver-reducing substance of the cells.

With Apáthy's and Bethe's definite results before us, as well as the less certain observations of many previous

[1] Bethe. "Ueber die Primitivfibrillen in den Ganglienzellen vom Menschen, &c." *Schwalbe's morphologische Arbeiten*, viii., 1898, p. 95.

[2] Golgi. "Sur la structure des cellules nerveuses." *Archives ital. de biol.*, xxx., 1898, p. 60.

observers, it is impossible to doubt that the non-staining substance of Nissl is traversed by fibrils, although we still have much to learn with regard to their thickness, course and disposition. Especially must we regard it as uncertain at present (1) whether the fibrils form a plexus within the cell or cross one another in a feltwork, and (2) whether each fibril is a distinct unit which always retains its individuality, or whether it may fuse with other fibrils. Bethe regards every fibril as an independent conducting strand, presumably incapable of fusion with other fibrils. Apáthy sees the fibrils unite to form thicker fibrils and subdivide into finer fibrils.

Accepting Bethe's description of fibrils, it is to be noted that he observes them entering and leaving the dendrites by their *branches* only. Here I venture to think that Bethe's observations are incomplete. It appears to me that no account of the minute anatomy of the nerve-cell is satisfactory which ignores the "thorns." Thorns as shown by chrome-silver colouration are most definite structures of obvious physiological importance. From Bethe's figures and description I gather that his staining method does not reveal their presence, and this being the case, I take it that his stain fills in a part of the picture only. The arguments already used lead me to the conclusion that *every thorn is a fibril near its junction with a cell.* Apáthy's and Bethe's "primitive fibrils" may be bundles of fibrils, or they may be fibrils formed by the fusion of a number of truly primitive fibrils—"elementary fibrils," as Apáthy terms them—but the combined fibrils are not properly entitled to be styled primitive. Every thorn is the cell-end of a primitive fibril. Probably the true primitive fibrils fuse to form the fibrils which Apáthy recognises, when stained with chloride of gold, in the same way as Apáthy's elementary fibrils unite into his "primitive fibrils," and then fuse again to form "neurofibrils."

In one of the papers already referred to I have hazarded the conjecture that nerve-conduction depends upon the establishment of a certain relation between the conducting fibrils and the cell-protoplasm by which they are invested.

I have endeavoured to show that if this be the case, all the phenomena of attention, inhibition, sleep, which have been supposed to have their physical basis in the condition of extension, to a greater or less extent, of pseudopodial processes of the dendrites, might be just as well explained as the result of the extension along the conducting fibrils, to a greater or less extent, of the cytoplasm. My hypothesis, indeed, removes a fundamental difficulty in the conduction-by-contact theory. Upholders of this theory speak of the extension of processes from one neuron to another as a natural physiological precursor of conduction. They talk of "conduction by contact" as if it presented no physical difficulties. They forget that at the time when an impulse has traversed the neuron A on its way to the neuron B, the approximation of the conducting processes of the two neurons can be effected by no means other than the *passage across the gap* which separates them of the impulse which is about to be conducted along a physiologically continuous strand. *Conductio in distans* must precede conduction by contact. In other words, the contact theory does not in any way explain the antecedent adjustment by which contact is brought about. Now, my hypothesis of continuity of the conducting filaments, with variability of their effectiveness as conductors, gives a more intelligible explanation of "attention" and "inhibition." The conducting fibrils never lose a low degree of conductivity. The first impulse which struggles through from nerve-cell A to nerve-cell B brings about an overflow of cytoplasm from the one to the other which, *ipso facto*, creates an open road along which subsequent impulses—all impulses are, of course, vibratory—pass with ease. At the same time other paths, not in the circuit, are blocked by withdrawal of cytoplasm; producing the well-known phenomenon of inhibition for all competing reflexes.

This hypothesis also disposes of what has been termed the "physiological evidence in favour of the neuron theory." Physiologists have resisted the doctrine of the continuity of conducting fibrils, on the ground that such continuity leaves the nerve-cells nothing to do—gives no opportunity for the

explanation of the tiring of nerve-cells, the delay of impulses, &c. But if effectiveness of conduction depends upon a certain relation between the conductor and the cytoplasm in which it is embedded, it is free to the physiologists to restore to the nerve-cells any prerogatives which they may consider that their status in the system requires.

We can imagine that some day the quality of all nerve phenomena, from a knee-jerk to a thought, will be explained as due to the distribution of sensory impressions through the labyrinth of fibrils, the conductivity of which is automatically regulated by the impulses themselves, within the limits imposed by the hereditary tendency of the individual's cytoplasm to overflow in certain directions, and with certain urgency, and the functional condition of the cytoplasm at the time; but until my hypothesis has some basis on histological observations it is futile to consider the more detailed applications which may be made of it.

Lastly, not all kinds of cells bear thorns. In the case of the cerebellum, for example, it is remarkable that whereas the superficial molecular cells, Cajal's cells, and Purkinje's cells are conspicuous for their thorniness, the cells of Golgi have thornless dendrites. As the cells of Golgi are indubitably nervous, the absence of thorns cannot be without significance; and one naturally notes that these cells are remarkable for the extreme richness of the branching of their axis-cylinders. Their elaborate arborisation provides a complicated system of intercommunication with other nerve-cells or fibres, without any need for the dendrite system to be called into play, to the same extent as in the cells of Purkinje for example. We may suppose that, in the case of Golgi's cells, the collection of fibrils from afferent nerves does not, as in the case of other cerebellar cells, occur chiefly through the dendrites. Hence the dendrites are not studded with the ends of entering fibrils, *i.e.*, there are no thorns.

The Connection of Dendrites with Connective Tissue Elements.

As is well-known the continuity of dendrites with pia mater and with blood-vessels was described by Golgi and

Rámon y Cajal. If the chrome-silver method is to be trusted such a connection is beyond dispute. The apical processes of developing granules are seen to end in knobs beneath the pia mater, and the connection of dendrites with connective tissue elements is in certain cases unmistakable. The dismissal of such connections as occasional does nothing to clear the ground. If it ever obtains, and it is difficult to suppose that the definite silver colouration occurs in the absence of any underlying substance, it is a matter of profound significance, for it compels the conclusion that the dendrites are not solely composed of conducting substance. They must consist of a non-conducting cell-substance which fuses with, or at any rate reaches to, the connective tissue elements, and of conducting substance, presumably fibrils, which does not extend to the tips of the dendrites. We must figure the cytoplasm as forming dendrites through which conducting fibrils pass. Provided the fibrils are isolated from the connective tissue it matters little whether the rest of the cell substance is connected with it or no. Rohde[1] described the continuity of the spongioplasm of nerve-cells with the spongioplasm of connective tissue cells in the electric lobe of Torpedo, whereas the hyaloplasm of the nerve-cell is distinct. He had not observed primitive fibrils.

Summary.

The neuron theory, or statement, alleges the anatomical independence of nerve-cells.

This statement is based upon appearances obtained by methods which colour the cytoplasm and leave the conducting elements uncoloured.

(1) Even in chrome-silver preparations evidence is forthcoming that fibres which start as the axons of certain larger cells end, after subdivision, by uniting directly with smaller cells. Such a connection of cell with cell is to be seen or inferred in the case of (*A*) the granules of the cerebellum and in (*B*) of the cells of the sympathetic system.

(*A*) If the granules of the cerebellum are observed in

[1] Rohde. *Arch. f. mikros. Anat.*, 1895, p. 408.

their embryonic condition—when they are carrot-like and as yet unfurnished with clawed arms—they are seen to be directly connected with fibres from the arbor vitæ. (*a*) The same fibre from the arbor vitæ may divide to form the axons of two or more granules. (*b*) A granule which gives the characteristic divaricating axon to the molecular layer may, in addition, send an axon to the arbor vitæ.

(*B*) There are reasons for concluding that the slender processes of cells of the sympathetic system (which are universally regarded as dendrites) are in reality axons or nerve-fibres. If this be correct each cell receives the axon of a cell higher in the system and gives off a bunch of finer axons for cells lower in the system—is, in fact, a distributing cell.

(2) Thorns are in no sense artifacts, but primitive fibrils invested by cytoplasm for a certain distance from the dendrites into which they enter. Since the cytoplasm is continued but a short distance along the fibrils, and since this substance is alone coloured by the methylene-blue or the chrome-silver methods, the cell-end of the fibril appears as a thorn, or rather as a rod with a knob (gemmule), owing to the semi-fluid sheath of cytoplasm having collected at its end into a droplet.

DESCRIPTION OF FIGURES.

In most of the figures the positions of the cells of Purkinje and of the fibres of the arbor vitæ are indicated. Their size and the distance between them shows the magnification.

PLATE 1.

FIG. 1.—Puppy, 6 days old. Cerebellum. Hardened in bichromate of potassium and osmic acid mixture for more than a year. Two granules with axis-cylinder processes which divaricate in the molecular layer and, in addition, slender processes passing to the arbor vitæ which have the appearance of axis-cylinders.

FIG. 2.—Kitten, 4 days old. Cerebellum. Hardened six months. A granule giving an axis-cylinder to the molecular layer and another to the arbor vitæ.

FIG. 3.—Same preparation as fig. 1. The fibre to the arbor vitæ gives off branches at right angles.

FIG. 4.—Same preparation as fig. 2. The fibre to the arbor vitæ is not so directly continuous with the granule as in figs. 1 and 2. It looks as if some sort of basket or "claw" connection were being formed.

FIG. 5.—Same preparation as fig. 2. Shows the same method of connection of a fibre from the arbor vitæ with a granule.

PLATE I.

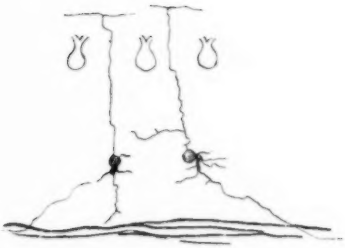

FIG. 1.

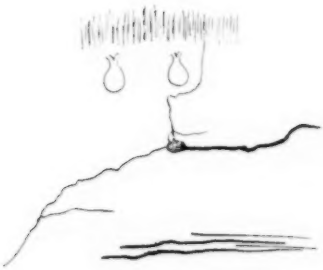

FIG. 2.

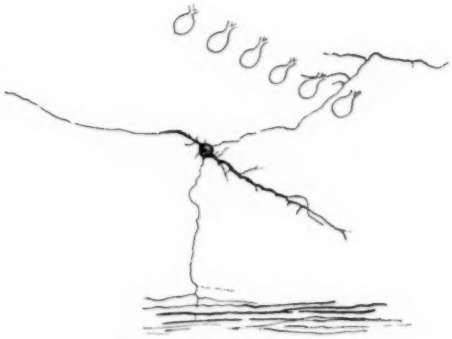

FIG. 3.

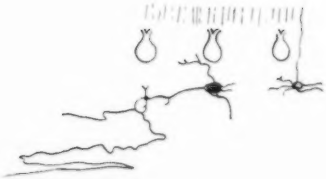

FIG. 4.

FIG. 5.

PLATE II.

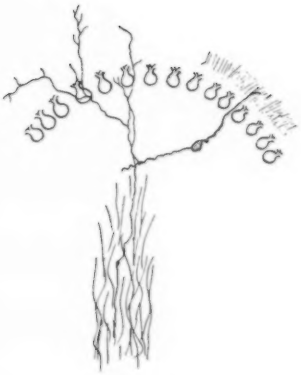

FIG. 6.

FIG. 7.

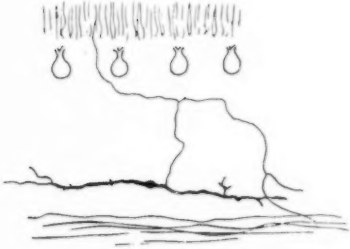

FIG. 8.

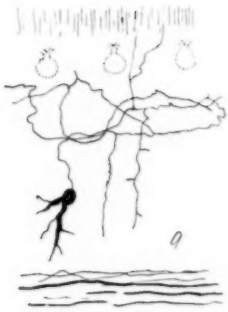

FIG. 9.

FIG. 10.

PLATE III.

FIG. 11.

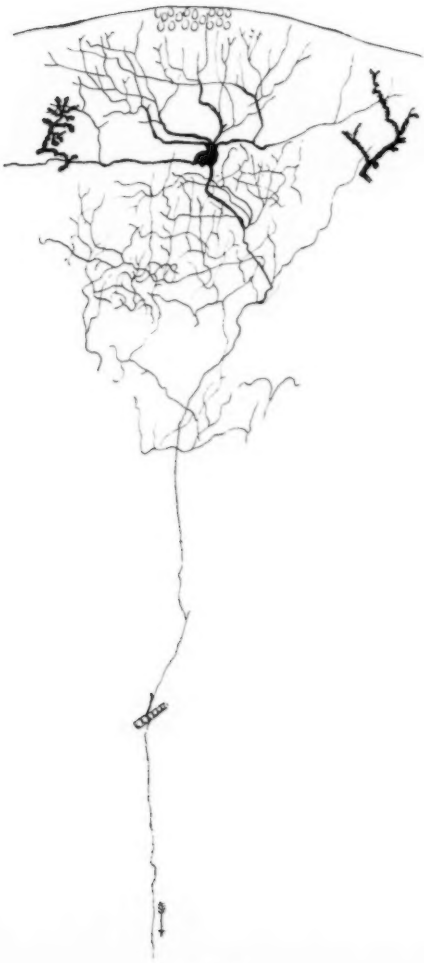

FIG. 12.

PLATE IV.

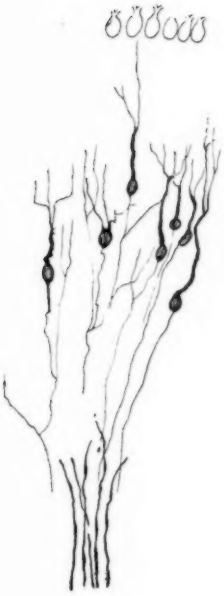

FIG. 13.

FIG. 14.

FIG. 15.

FIG 16.

FIG. 17.

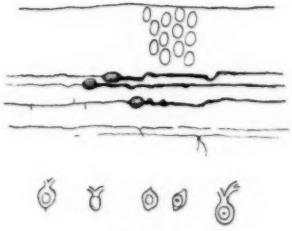

FIG. 18.

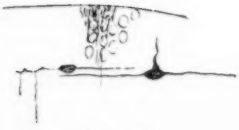

FIG. 19.

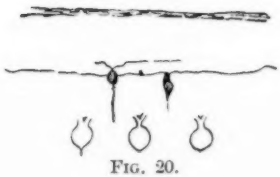

FIG. 20.

PLATE V.

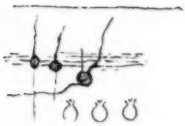

FIG. 21.

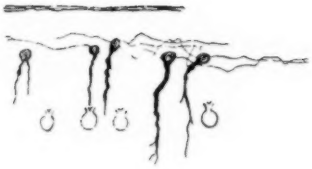

FIG. 22.

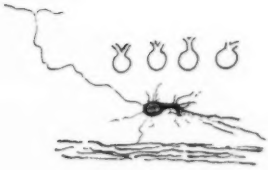

FIG. 23.

FIG. 24.

FIG. 25.

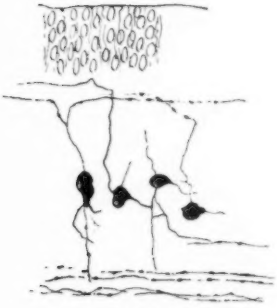

FIG. 26.

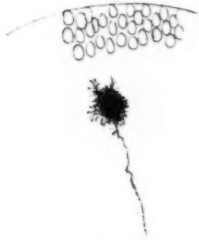

FIG. 27.

FIG. 28.

FIG. 29.

PLATE 2.

FIG. 6.—Same preparation as fig. 2. A granule which shows a curiously thick process passing between the cells of Purkinje into the molecular layer, and a centripetal axon which appears to join a fibre which branches in the molecular layer.

FIG. 7.—Same preparation as fig. 2. The axon of the granule divides into one branch which passes backwards to the arbor vitæ and another branch which again gives off a branch before it enters the molecular layer.

FIG. 8.—Rat, 12 days old. Cerebellum hardened in bichromate of potassium and osmic acid for twelve months. The granule, which lies near to and parallel with the fibres of the arbor vitæ, gives off an axon which divaricates before it has passed the layer of cells of Purkinje, one of the two branches entering the molecular layer and the other returning towards the arbor vitæ.

FIG. 9.—Kitten, 10 days old. Hardened six months. A granule, the axon of which has a very complicated and tortuous course before it gives a branch which bifurcates on its way to the molecular layer.

FIG. 10.—Kitten, 4 days old. Two carrot-granules at opposite sides of the folium are provided with axons which unite into a single fibre which can be followed for a considerable distance in the arbor vitæ. The nuclei of Obersteiner's layer are shown at one place beneath the pia mater.

PLATE 3.

FIG. 11.—Kitten, 7 days old. Hardened for two months. Two granules with united axons.

FIG. 12.—A single Golgi-cell is seen lying between two fragments of the thorny dendritic systems of Purkinje-cells at the apex of a folium. The dendrites of the Golgi-cell are thornless. The axon of the cell, after giving rise to a copious arborescence, is continued to the very base of the folium, a distance of at least 2 mm.

PLATE 4.

FIG. 13.—Kitten, 1½ days old. Hardened nine months. The section, which cuts the folium transversely, passes obliquely through the granular layer, from the cells of Purkinje to the arbor vitæ. It shows a very typical group of carrot-granules. The axons of some of the granules are unbranched, others give off collaterals.

FIG. 14.—Kitten, 7 days old. Hardened in bichromate of potassium and osmic acid for nearly two years. The cell, which is rather larger than an ordinary granule, gives off three processes, each of which has the character of an axon.

FIGS. 15, 16, 17.—Kitten, 4 days old. Three groups of cells placed vertically to the pia mater, which appear to be developing carrot-granules. Figs. 15 and 17 with obj. ⅓ in., oc. 4; fig. 16, obj. ⅛ in., oc. 8.

FIG. 18.—Kitten, 17 days old. Section parallel to long axis of folium; oblique to surface. In the molecular layer just below Obersteiner's layer (superficial granular layer) are three cells lying parallel to the surface. The distinction between the apical process and the axon is marked with unusual clearness; but every intermediate form is to be observed between such cells as these and cells which appear to give off a long axis-cylinder process on either side. The axon of the deepest of the three cells gives short lateral branches. The divaricating fibres into which the axons of granules divide are seen beneath the three "fibre-cells."

FIG. 19.—Kitten, 17 days old. Two tangential cells in the molecular layer, immediately beneath Obersteiner's layer. The cell to the right shows an apical process directed towards the surface.

FIG. 20.—Kitten, 17 days old. Two granules in an early stage of development. They lie in the molecular layer just beneath Obersteiner's nuclei. According to Rámon y Cajal's account they have been formed by the pinching together of the processes of tangential cells similar to those shown in figs. 18 and 19.

PLATE 5.

FIG. 21.—Kitten, 4 days old. Cells in molecular layer just beneath tangential cells of an ambiguous character—apparently developing carrot-granules.

FIG. 22.—Kitten, 1½ days old. Developing granules the apical processes of which reach as far as, or beyond, the cells of Purkinje.

FIG. 23.—Puppy, 6 days old. A granule in a more advanced condition. The apical process is carrot-shaped with large numbers of slender branches. It lies almost parallel to the fibres of the arbor vitæ. Its axon bifurcates in the molecular layer.

FIG. 24.—Hedgehog, 2 weeks old. The apical process appears to join one of the branches of a "mossy" fibre. This is typical of a large number of preparations in which a direct union between a mossy fibre and the apical process of a developing granule can be traced.

FIG. 25.—Hedgehog, 2 weeks old. A granule which still shows an apical process extending into the arbor vitæ as well as an axon which enters the molecular layer; but it also exhibits commencing arms with claws. Figs. 20, 22, 23, 24, 25 represent successive stages in the development of granules, intermediate between the tangential "fibre-cell" and the adult round granule with five or six clawed arms.

FIG. 26.—Kitten, 2 days old. Four granules which lie in the granular layer. Each gives a characteristic axon to the molecular layer, and, in addition, several slender processes towards the arbor vitæ. The processes of the two middle granules appear to unite together.

FIG. 27.—Kitten, 1½ days old. A young Purkinje-cell which has not yet developed a dendritic system. A multitude of fibrils appear to enter the body of the cell.

FIG. 28.—Hedgehog, between 2 and 3 weeks old. A young Purkinje-cell, the dendritic system of which has begun to develop. The body of the cell has fewer fibrils connected with it than in fig. 27.

FIG. 29.—Puppy, 6 days old. The dendritic system is more developed than in fig. 28. The body of the cell is almost free from fibrils. A somewhat more advanced Purkinje-cell. The thorns in the last two specimens are much larger and coarser than they are in the adult.

A CASE OF VIVID AFTER-IMAGES EXPLAINED ON HERING'S THEORY.

BY DAVID FRASER HARRIS, M.D., B.SC.(LOND).

Lecturer on Physiology in the University of St. Andrews.

THE phenomena recorded and analysed below were experienced by myself when I was occupied in estimating the principal focal lengths of one or two convex lenses.

While focussing the sun's rays at noon upon a sheet of white paper, and without at the moment thinking of the probable effect on the retina, I stared at the dazzlingly white spot for some time, until roused by noticing that the paper was on the point of being set on fire.

On turning my eyes towards the least well-lighted corner of the white-walled room, I noticed a most pronounced black after-image, which persisted for about thirty seconds. Had this merely faded away and given place to normal colour-vision, there would have been nothing more to be experienced than the familiar sensation of being "blinded" as after gazing at the sun itself; but the black patch was followed by two coloured images of such brilliance and, *pro tem.*, apparently so persistent, that a record of them may be pardoned.

The first was a beautiful rose-pink patch, referable to the part of the retina concerned in direct vision; it lasted about three minutes; it was replaced by a brilliant canary-yellow patch of rather larger size, persisting for about twice as long. Before this last had faded I had to leave the building, and so experienced the very unusual sensation of seeing, almost as through a piece of yellow glass, all objects projected on the central portion of the retina; while at the same time their colours seemed less changed than if yellow glass had really been used.

It seems to me that Hering's theory of colour-vision accounts for the colours of these after-images, and for their order of succession.

The theory virtually involves the following (for light of ordinary intensities) :—

(1) That white light katabolises the white-black substance,[1] and the vision of a black object (or darkness) anabolises it; but further, that each coloured ray, *i.e.*, of homogeneous light, katabolises the W.-B. Sb., so that whatever other (chromatic) effects it may have, each *coloured* light gives rise to a certain amount of the sensation of white—to a very different degree for the various rays, yellow doing so most of all—hence, as Foster[2] remarks, "a fully saturated colour . . . . . according to this view does not exist."

(2) That katabolism of the red-green substance[3] gives rise to a sensation of red, anabolism of it, of green; that if there is metabolic equilibrium (as under the influence of spectral yellow) there is neither red nor green since opposite tendencies are neutralised, but that if any katabolism predominates over any co-existing anabolism, the sensation will tend towards "orange"; if anabolism predominates, the sensation will tend towards a greenish hue.

(3) Similarly for the yellow-blue substance;[4] katabolism alone gives yellow, anabolism blue, a predominance of the one a yellowish, of the other a bluish hue; while spectral green leaves it in metabolic equilibrium.

Thus the various homogeneous constituents of heterogeneous (white) light are supposed to simultaneously affect the several hypothetical visual substances in such a fashion that the tendencies to katabolism are just balanced by those to anabolism, and hence no *chromatic* sensations result—at least not with diffuse light of ordinary intensity; but it must be somewhat different with sunlight of such unusual brilliance as to temporarily "blind" the retina (produce the black negative after-image).

With such light there are, in fact, present the conditions to ensure, on the whole, a marked excess of katabolism over anabolism, and seeing that in noonday sunlight there is a relative predominance of such rays as are associated with maximal luminosity, viz., the orange-yellow, the first effect in consciousness would be a sensation of white so intense as to render for a time any concomitant chromatic sensations unperceived.

Violent katabolism has been set up in the R.-G. and Y.-B. substances, and will make itself felt in consciousness as soon as the negative after-image of black (resulting from the rapid reconstitution of the W.-B. Sb. on the cessation of the very violent stimulation) has passed away.

[1] W.-B. Sb. [2] "Text-book of Physiology," part iv., 6th ed., p. 1349.
[3] R.-G. Sb. [4] Y.-B. Sb.

The first effect is that of a rose-pink patch from the katabolism of the R.-G. Sb. by the orange rays, virtually a positive after-effect due to retino-cerebral functional inertia. It is pink and not a pure or saturated red because, *ex-hypothesi*, there is always some concomitant katabolism of the W.-B. Sb. along with any effect due to coloured rays. As soon, however, as the metabolic equilibrium of the R.-G. Sb. has been re-established, a yellow patch is perceived due to the predominance of katabolism over anabolism in the Y.-B. Sb. We have experimental evidence[1] that, as studied under appropriate conditions, the sensation of red is elicited in a shorter time than that of green, and still more so than that of violet; in other words, yellow takes a longer time to be developed than red.

Thus, as the red sensation passes off, the yellow is reaching its maximum,[2] the katabolic phase in the Y.-B. Sb. long out-living the period of action of the stimulus which produced the disturbance. The yellow is now the only chromatic sensation not neutralised, and it is perceived in its fulness until, on the metabolic equilibrium being re-established, it, too, fades away, and the retina recovers from its state of over-stimulation.

Review.

Neurypnology, or the Rationale of Nervous Sleep, considered in Relation to Animal Magnetism or Mesmerism and Illustrated by Numerous Cases of its Successful Application in the Relief and Cure of Disease. By JAMES BRAID, M.R.C.S., C.M.W.S., &c. A new edition, edited with an introduction, biographical and bibliographical, embodying the author's later views and further evidence on the subject, by ARTHUR EDWARD WAITE. London, George Redway. 1899.

THE above work comprises (1) a biographical introduction, which gives a short account of Braid's life and a more extended one of his writings; (2) a reproduction of the original edition of "Neurypnology," which forms the greater part of the volume; (3) an appendix of editorial notes chiefly drawn from Braid's later works; and (4) a bibliography of Braid's writings.

According to Mr. Waite, little is known about Braid in this country, and there is no trustworthy bibliography of his writings.

[1] Stewart, *P.R.S.E.*, 1888, p. 441.
[2] It has a slightly greater degree of functional inertia.

His later works, rare and almost unknown, have been traced with difficulty. Mr. Waite, however, claims to have been successful in his researches, and presents, as their result, a work on Braid which he asserts to be substantially complete.

Mr. Waite has undoubtedly rendered a service to hypnotic literature, but he has not made the most of the material collected, and his work is neither as complete nor as accurate as he believes it to be. The following are a few, amongst many, objections which might be urged against it: Thus, we are told that the discovery of hypnotism alone lifted Braid out of the groove of the ordinary practitioner. This is incorrect. Before he discovered hypnotism Braid had operated on 262 cases of talipes, 700 cases of strabismus, and twenty-three cases of spinal curvature, and his reputation as a surgeon attracted patients from all parts of the country.

Although "Neurypnology" is historically interesting, it must not be forgotten that it was written almost immediately after Braid commenced hypnotic work, and that later his views underwent a complete change. The French translation of "Neurypnology" is more valuable than Mr. Waite's, as in an appendix of thirty-six pages it gives a full translation of Braid s last manuscript, containing a summary of his more matured theories. This is barely referred to by Mr. Waite.

Some passages show a somewhat unusual want of care in compilation. Thus (pages 317-319), a case of torticollis cured by suggestion is recorded at length, and we are told that a year later there had been no relapse. The case is repeated (pages 343, 344) along with the fresh statement that "whether the cure was permanent does not appear from the narrative."

The bibliography, to which Mr. Waite attaches great importance, only imperfectly reproduces those I myself published at earlier dates (BRAIN, part lxxiii., Spring, 1896; *Proceedings of the Society for Psychical Research*, part xxx., June, 1896; *Revue de l'Hypnotisme*, June, 1898). In seven of the works referred to by Mr. Waite the title is wrong or incomplete; in twenty-one, quoted from medical journals, neither volume nor page is given. The only new material is two unimportant letters in reply to one from "James Simpson, Esq., advocate," erroneously described by Mr. Waite as "James Singer," while in my bibliographies are to be found nine of Braid's writings which are omitted from Mr. Waite's. Further, some of Braid's works, of which Mr. Waite has been unable to trace a single copy, have long been in my possession.

According to Mr. Waite, Braid's connection with the debated subject of phrenology terminated with the phreno-hypnotic experiments recorded in "Neurypnology." The subject, we are told, remains where he left it, while Dr. Foveau de Courmelle's verdict, viz., that the phenomena are as likely to be the result of real action as of unconscious suggestion, is accepted by the unprejudiced. Mr. Waite is ignorant of, or ignores, Braid's later

experiments. From these Braid concluded that all phrenological phenomena were the result of direct or indirect suggestion, and arose either from a previous knowledge of phrenology or from a system of training during hypnosis. They manifested themselves when points were touched, which audible suggestion had taught the subjects to associate with particular ideas. Further, pressure upon the head was not necessary, and the associations could be established in the same artificial way between the phenomena and other parts of the body. Finally, the excitation of the muscles of expression was capable of arousing corresponding ideas in the subject's mind.

It is certainly surprising to be told at the present day that the phenomena of phrenology are as likely as not to be genuine.

Mr. Waite's account of Braid's views on mesmerism or animal magnetism demand serious notice. Formerly, according to him, it was generally but erroneously believed that Braid opposed animal magnetism. Thanks, however, to French writers this error has been rectified. Braid was the champion and witness of animal magnetism, and placed the central fact of mesmerism beyond all further dispute. In opposition to this it is to be noted, and the point cannot be too strongly insisted upon, that the primary and cardinal fact on which Braid founded all his theories of hypnotism was the purely subjective origin of its phenomena. Everything depended on the physical and mental condition of the patient; not on the volition or passes of the operator which threw out magnetism, or called into action some occult fluid or medium. Braid never swerved from this view. Later, he repeated it more emphatically, and demonstrated experimentally that all the phenomena attributed to magnets, metals, drugs in sealed tubes, &c., could be equally well produced by direct or indirect suggestion when the supposed exciting agents were absent. Thus, a wooden magnet acted like a real one when the subject was deceived as to its nature, whereas a genuine one produced nothing if the subject did not know it was there.

According to Waite, "the metalo-therapeutic theory, first discovered by Burq and developed by Charcot, inaugurated a new era of hypnotism, and led to that long series of researches connected with the Salpêtrière School of Paris, which have done so much to obscure, if not, indeed, to tarnish, the rival lustre of the Nancy School of Liébault."

This view of metallo-therapeutics is many years too late. The opinions of Burq, which were shared by Elliotson and the mesmerists of his day, were successfully combated by Braid. Charcot simply revived an ancient fallacy, which the Nancy School, following in Braid's footsteps, again overthrew. As far back as the International Congress of Psychology of 1892, the Charcot theories had practically ceased to excite scientific interest.

The assertions of the mesmerists as to the clairvoyant powers of their subjects were described by Braid as opposed to all that was known of physical science. The following, he said, were

frequent sources of error in clairvoyant experiments:—(1) The hyperæsthesia of the organs of special sense, which enabled impressions to be perceived through the ordinary media that would pass unrecognised in the waking condition. (2) The docility and sympathy of the subjects, which tended to make them imitate the action of others. (3) The extraordinary revival of memory by which they could recall things long forgotten in the waking state. (4) The remarkable effect of contact in arousing memory. (5) The condition of double consciousness or double personality. (6) The vivid state of the imagination, which instantly invested every suggested idea, or remembrance of past impressions, with the attributes of present realities. (7) The tendency of the human mind, in those with a great love of the marvellous, to erroneously interpret the subject's replies in accordance with their own desires. (8) Deductions rapidly drawn by the subject from unintentional suggestions given by the operator.

According to Braid, the belief in thought-transference was also the result of careless experiment, and he had never met with a subject who could divine his thoughts when their transmission by the recognised media had been rigorously excluded.

Braid did not believe that mesmeric subjects possessed intuitive powers. Doubtlessly some of them successfully predicted their own hysterical attacks, but the prophecy simply produced its own fulfilment through self-suggestion.

According to Mr. Waite, "it has been rightly observed that Braid, the discoverer, so to speak, of suggestion, by no means realised the possibilities of suggestion of the unconscious kind." Bernheim, and other writers unacquainted with Braid's later works, had previously made similar statements. It is difficult, however, to understand how Mr. Waite could commit a similar error. Braid repeatedly, clearly, and emphatically pointed out that unconscious suggestion accounted for all the phenomena which the mesmerists attributed to magnets, metals, and occult fluids. Sometimes, he said, the suggestions were given in their most obvious form by the operator verbally predicting in the subject's hearing the nature of the phenomena which ought to appear. At others they were given less directly by passes, manipulations, excitation of the muscles of expression, &c.; but these owed their power, not to their physical influence, but to the ideas they excited in the subject's mind. When all forms of suggestion were rigorously excluded, the mesmeric phenomena did not appear. This, Braid asserted, proved the influence was a mental one; were it electric, suggestions were as unnecessary in the case of the subject as in that of a galvanic battery.

Apparently Mr. Waite believes in animal magnetism, metallo-therapeutics, phrenology, and clairvoyance. All these things Braid successfully combated, and to attribute to him any belief in their existence shows an absolute failure to grasp the spirit and significance of his teaching.

J. Milne Bramwell.

Rules

AND

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OF THE

Neurological Society of London,

1900.

WITH THE

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Neurological Society of London.

REPORT OF COUNCIL.

FEBRUARY 8, 1900.

The Council has much pleasure in reporting the continued success and prosperity of the Society. The members now number 216.

The Inaugural Address was delivered at the Annual General Meeting on February 2nd, 1899, by Dr. W. H. GASKELL, F.R.S., on "The Meaning of the Cranial Nerves."

Sir SAMUEL WILKS, Bart., F.R.S., the second President of the Society, has been elected an Honorary Member.

During the summer the Society met out of London for the second time since its constitution, and as on the former occasion, the Meeting was held in the Physiological Department at Cambridge, where Dr. RIVERS and Mr. MYERS made communications on the "Psychology of the Natives of Borneo and Torres Strait."

In response to an invitation from the Secretaries of the Section of Neurology at the Thirteenth International Congress of Medicine, to be held in Paris, in 1900, Dr. DAVID FERRIER, F.R.S., and Dr. C. S. SHERRINGTON, F.R.S., have been nominated to represent the Society at that Congress.

As has now become the custom, the Proceedings of the Society have been published at some length in "Brain," and an abstract appended to this Report shows the value and character of the work that has been done during the year.

Proceedings of the Society during the year 1899.

February 2nd, Dr. W. H. GASKELL, F.R.S., Inaugural Address, "The Meaning of the Cranial Nerves."

March 2nd, Dr. STANSFIELD, "Epileptic Insanity associated with Mastoid Disease; operation; improvement of mental symptoms." Dr. F. E. BATTEN, "Unusual Form of Muscular Atrophy in a Boy." Dr. CALEY, "Case with Symptoms of Syringo-myelia with Sudden Increase followed by Sudden Diminution in Intensity." Dr. WILFRED HARRIS, "Hæmorrhage into the Cord. Dr. J. H. BRYANT, "Tremors of Hand like those of Paralysis Agitans, following Typhoid Fever." Dr. TOOTH, "Peroneal Type of Muscular Atrophy."

May 11th, in the Physiological Laboratory at St. Mary's Hospital, Dr. WALLER, F.R.S., Demonstration of the Action of Veratrine, Betaine, Neurine, Choline, Muscarine, Snake Venom, and Tetanus Toxin on Isolated Nerve. Professor E. A. SCHÄFER, F.R.S., Microscopic Sections illustrating (1) Termination of the Fibres of the Pyramidal Tracts of the Cord; (2) Origin of the Fibres of the Dorsal and Ventral Cerebellar Tracts of the Cord.

June 17th, at the Physiological Laboratory, Cambridge, Dr. W. H. R. RIVERS and Mr. CHARLES S. MYERS, "Some Psychological Observations on the Natives of Torres Straits and Borneo."

October 26th, Dr. J. A. COUTTS and Dr. A. M. GOSSAGE, "Unusual Case of Paralysis of Acute Onset." Dr. JAMES COLLIER, "A Case with Acute Onset resembling Friedreich's Disease." Dr. JAMES COLLIER, "Case of Myopathy of Landouzy-Dejerine Type." Dr. WILFRED HARRIS, "Case of Myopathy of Landouzy-Dejerine Type." Dr. C. E. BEEVOR, "A Case of Cerebral Degeneration (Diplegia)."

December 7th, Dr. F. E. BATTEN and Dr. J. S. COLLIER, "Spinal Cord Changes in Cases of Cerebral Tumour."

December 21st (Adjourned Meeting), Mr. VICTOR HORSLEY, F.R.S., "Brown-Séquard Fits in a Cat." Dr. WILFRED HARRIS, "An Experimental Investigation on the Decussation of the Optic Nerves in various Animals, and on the Path of the Reflex for the Contraction of the Pupil to Light."

BALANCE SHEET, 1899.

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Receipts.	£	s.	d.
To Balance	65	16	2
Subscriptions	200	6	0
Half Share of profit on "Brain" ...	54	8	4
Contribution by Messrs. Macmillan towards cost of Illustrations in "Brain"	18	0	0
	£338	10	6

Expenditure.	£	s.	d.	£	s.	d.
By Stationery and Printing—						
Pulman & Son	2	3	0			
Bale & Danielsson ...	16	5	11			
				18	8	11
Illustrations—Bale & Danielsson ...				124	19	6
Microscopes—Baker				4	2	0
Hire of Rooms and Refreshments—Medical Society				19	15	0
Messrs. Macmillan for publishing "Brain"				124	6	0
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Balance				45	9	2
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Audited and found correct December 6, 1899,

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RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due, shall be considered as equivalent to resignation.

15.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

16.—Five Members of the Council shall form a quorum.

17.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

18.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

19.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

20.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

21.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

22.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

23.—There shall be six Ordinary Meetings annually, of which the first held in each year shall be the Annual General Meeting.

24.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

25.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

26.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

27.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

28.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

29.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watteville, M.A., M.D., B.Sc.

HONORARY MEMBERS.

Mr. Herbert Spencer, London.
Professor Wundt, Leipzig.
„ Hitzig, Halle.
Sir Samuel Wilks, Bart., London.

CORRESPONDING MEMBERS.

Professor Erb, Heidelberg.
„ Golgi, Pavia.
„ Goltz, Strasburg.
„ Grasset, Montpellier.
„ Jolly, Berlin.
„ Luciani, Rome.
„ Munk, Berlin.
„ Obersteiner, Vienna.
„ Retzius, Gustaf, Stockholm.
„ Ribot, Paris.
Dr. Weir Mitchell, Philadelphia.

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886 J. Hughlings Jackson, M.D., F.R.S.
1887 Sir Samuel Wilks, Bart., M.D., F.R.S.
1888 Sir James Crichton-Browne, M.D., F.R.S.
1889 Jonathan Hutchinson, F.R.S.
1890 Thomas Buzzard, M.D.
1891 John S. Bristowe, M.D., F.R.S.
1892 H. Charlton Bastian, M.D., F.R.S.
1893 Edward A. Schäfer, F.R.S.
1894 David Ferrier, M.D., F.R.S.
1895 Sir William H. Broadbent, Bart., M.D., F.R.S.
1896 Alexander Hill, M.A., M.D.
1897 George H. Savage, M.D.
1898 Victor Horsley, M.B., F.R.C.S., F.R.S.
1899 W. H. Gaskell, M.D., F.R.S.
1900 Augustus Waller, M.D., F.R.S.

LIST OF HUGHLINGS JACKSON LECTURERS.

1897 J. Hughlings Jackson, M.D., F.R.S.

*** Members are requested to communicate with the Secretaries when corrections are necessary.

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EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.	Tr., Treasurer.
Pres., President.	Sec., Secretary.
V.P., Vice-President.	C., Member of Council.

H.J.L., Hughlings Jackson Lecturer.

ORDINARY MEMBERS.

Elected

1899 ABRAHAMS, BERTRAM, M.B., M.R.C.P., 14, Welbeck Street, W.

1892 ALEXANDER, ROBERT REID, M.D., The Asylum, Hanwell, W.

1897 ANDERSON, H. KERR, M.B., B.Sc., 9, Park Terrace, Cambridge.

1894 ANDRIEZEN, W. LLOYD, M.D., 7, Apsley Terrace, Acton, W.

1891 BALLANCE, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897-1900.)

O.M. BARLOW, THOMAS, M.D., F.R.C.P., 10, Wimpole Street, W.

1897 BARRATT, J. O. WAKELIN, M.D., F.R.C.S., Pathological Laboratory, West Riding Asylum, Wakefield.

O.M. BASTIAN, H. CHARLTON, M.D., F.R.C.P., F.R.S., 8A, Manchester Square, W. (Pres., 1892; V.P., 1891: C., 1886-89, 1893-95.)

1897 BATTEN, FREDERICK E., M.D., M.R.C.P., 124, Harley Street, W.

O.M. BEACH, FLETCHER, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey.

,, BEEVOR, C. E., M.D., F.R C.P., 33, Harley Street, W. (Tr., 1894-1900; C., 1893.)

1888 BIRT, ERNEST, M.D., L.R.C.P., M.R.C.S., West Riding Asylum, Wakefield.

1898 BOLTON, JOSEPH SHAW, M.D., B.S., B.Sc., Claybury Hall, Woodford, Essex.

1896 BOND, C. HUBERT, M.D., D.Sc., London County Asylum, Bexley, Kent.

1899 BOND, J. W., M.D., B.S.Lond., 26, Harley Street, W.

1892 BOWLBY, ANTHONY A., F.R.C.S., 24, Manchester Square, W.

1898 BOYCE, RUBERT W., M.B., University College, Liverpool.

,, BOYCOTT, ARTHUR NORMAN, M.D., Hertfordshire County Asylum, Hill End, St. Albans.

,, BRADBURY, JOHN B., M.D., F.R.C.P.Lond., 4, St. Peter's Terrace, Cambridge.

1889 BRADFORD, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 8, Manchester Square, W.

Elected

O.M. BRAMWELL, BYROM, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894.)

1899 BRAMWELL, EDWIN, M.B., C.M., National Hospital, Queen Square, W.C.

1895 BRAMWELL, JOHN MILNE, M.B., C.M., 2, Henrietta Street, W.

1892 BRISTOWE, HUBERT CARPENTER, M.D., L.R.C.P., M.R.C.S., 18, Warwick Road, Maida Vale.

O.M. BROADBENT, Sir WM. H., Bart., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.P., 1893; C., 1886-87, 1896.)

1894 BRODIE, T. GREGOR, M.D., L.R.C.P., Lindfield, Uxbridge Road, Surbiton.

1897 BROWN, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 BROWN, SANGER, M.D., 100, State Street, Chicago.

O.M. BRUCE, ALEX., M.D., F.R.C.P.Ed., 13, Alva Street, Edinburgh. (C., 1895.)

,, BRUCE, J. MITCHELL, M.D., F.R.C.P., 23, Harley Street, W.

1898 BRUCE, SAMUEL NOBLE, M.R.C.S., 43, Kensington Gardens Square.

O.M. BRUNTON, Sir T. LAUDER, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896-98.)

1895 BRYAN, FREDERICK, M.B., M.R.C.P., The Asylum, New Southgate, N.

,, BRYANT, JOHN HENRY, M.D., L.R.C.P., 8, St. Thomas St., S.E.

1890 BURY, JUDSON SYKES, M.D., F.R.C.P., 10, St. John Street, Manchester.

O.M. BUZZARD, THOMAS, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-89; C., 1886-87, 1891-95.)

1898 CALEY, ALBERT, M.D., M.R.C.P., 24, Upper Berkeley Street, W.

,, CAMPBELL, HARRY, M.D., B.S., F.R.C.P., 23, Wimpole Street, W.

1887 CATTELL, J. McK., M.A., Ph.D., Columbia College, New York, U.S.A.

1899 CHAMBERS, JAMES, M.A., M.D., The Priory, Roehampton.

1891 CLARKE, J. MICHELL, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol.

1898 CLINCH, THOMAS ALDOUS, M.D., Durham County Asylum, Winterton, Ferryhill, Co. Durham.

O.M. COBBOLD, C. S. W., M.D., F.R.C.P.Ed., Bailbrook House, Bath.

1892 COLE, ROBERT HENRY, M.D., M.R.C.P., M.R.C.S., 53, Upper Berkeley Street, Portman Square.

1890 COLMAN, W. S., M.D., F.R.C.P., 22, Wimpole Street, W. (Sec., 1899-1900.)

,, CORNER, HARRY, M.D., L.R.C.P., Brook House, Southgate, Middlesex.

1899 COUPLAND, SIDNEY, M.D., F.R.C.P.Lond., 16, Queen Anne Street, W.

1895 DAVIDSON, ANDREW, M.B., C.M., The County Asylum, Dorchester, Dorset.

1897 DAWSON, WILLIAM R., M.B., B.Ch., Farnham House, Finglas, Co. Dublin, Ireland.

Elected

1894 DEAN, H. PERCY, M.D., F.R.C.S., 69, Harley Street, W.

1898 DODD, HENRY WORK, F.R.C.S., 136, Harley Street, W.

O.M. DRESCHFIELD, JULIUS, M.D., F.R.C.P., 325, Oxford Road, Manchester. (V.P., 1898-99.)

,, DRUMMOND, DAVID, M.D., 6, Saville Place, Newcastle-on-Tyne.

1892 DUPUY, EUGENE, M.D., 53, Avenue Montaigne, Paris.

1889 ECCLES, A. SYMONS, M.B., M.R.C.S., 23, Hertford Street, Mayfair, W.

1899 FAWCETT, JOHN, M.D., M.R.C.P., F.R.C.S., 24, St. Thomas Street, S.E.

O.M. FERRIER, DAVID, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1894; V.P., 1892-93; C., 1886-90-95.)

,, FOX, E. LONG, M.D., F.R.C.P., Church House, Clifton, Bristol.

,, FOSTER, Sir MICHAEL, M.D., F.R.S., Shelford, Cambridge.

,, GASKELL, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (Pres., 1899; V.P., 1897-98; C., 1896, 1900.)

,, GEE, SAMUEL J., M.D., F.R.C.P., 31, Upper Brook Street, W.

,, GODLEE, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1899 GOODALL, EDWIN, M.D., M.R.C.P., Joint Counties Asylum, Carmarthen.

1898 GOODLIFFE, JOHN HENRY, M.D. Aberd., County Asylum, Berry Wood, Northampton.

1897 GOSSAGE, ALFRED MILNE, M.B., M.R.C.P., 54, Upper Berkeley Street, W.

1892 GOTCH, FRANCIS, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 GRANT, DUNDAS, M.D., F.R.C.S., 8, Upper Wimpole Street, W.

O.M. GUNN, R. MARCUS, M.B., F.R.C.S., 54, Queen Anne Street, W. (C., 1899-1900.)

1895 GUTHRIE, LEONARD, M.D., M.R.C.P., 15, Upper Berkeley Street, W.

1893 HALLIBURTON, WILLIAM DOBINSON, M.D., F.R.C.P., F.R.S., Church Cottage, 17, Marylebone Road, N.W.

O.M. HAMILTON, D. J., M.B., F.R.S.E., University, Aberdeen.

1898 HANBURY, WILLIAM R., M.R.C.S., L.R.C.P., 27, Station Road, Watford.

1897 HARRIS, WILFRED J., M.D., M.R.C.P., 19, Oxford Terrace, W.

1898 HARTLEY, W. DARLEY, M.D., M.R.C.P., "Mayfield," Gilmore Hill Road, Cape Town, South Africa.

,, HASLETT, JOHN HANFIELD, M.R.C.S., L.R.C.P., Halliford House, Sunbury-on-Thames.

1891 HAWKINS, HERBERT PENNELL, M.D., F.R.C.P., 56, Portland Place, W.

O.M. HAYCRAFT, J. B., M.B., F.R.S.E., The University, Cardiff.

1891 HEAD, HENRY, M.D., M.R.C.P., F.R.S., 61, Wimpole Street, W. (C., 1900.)

O.M. HILL, ALEX., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.P., 1895; C., 1892-94.)

Elected

O.M. HOPKINS, JOHN, F.R.C.S., London Sick Asylum, Cleveland Street, W.

,, HORSLEY, VICTOR, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.P., 1896-97; C., 1892-93, 1899.)

1889 HUGGARD, WILLIAM R., M.D., F.R.C.P., Davoz Platz, Switzerland.

1896 HULME, GEORGE FREDERICK, M.D., C.M., Montague Road, Felixstowe.

1889 HUMPHRY, LAURENCE, M.D., M.R.C.P., 3, Trinity Street, Cambridge.

O.M. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.P., 1887-88; C., 1886, 1890.)

1889 HYSLOP, T. B., M.D., C.M., Bethlem Royal Hospital, Lambeth, S.E.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887-92.)

,, JESSOP, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 JOHNSTON, GEORGE, M.D., M.R.C.P., 3, Montagu Place, Montagu Square, W.

1892 JONES, HENRY LEWIS, M.D., F.R.C.P., 61, Wimpole Street, W.

1894 JONES, ROBERT, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

,, KIDD, PERCY, M.D., F.R.C.P., 60, Brook Street, W.

1896 LANGDON, F. W., M.D., Cincinnati, Ohio, U.S.A.

1898 LANGDON-DOWN, REGINALD L., M.B., B.Ch., M.R.C.P., 81, Harley Street, W.

O.M. LANGLEY, J. N., M.A., D.Sc., F.R.S., Trinity College, Cambridge. (C., 1898-99.)

1888 LAWFORD, J. B., M.D., F.R.C.S., 99, Harley Street, W.

O.M. LEES, D. B., M.D., F.R.C.P., 22, Weymouth Street, W. (C., 1900.)

1897 LEWIS, H. WOLSELEY, M.R.C.S., L.R.C.P., Claybury Asylum, Woodford, Essex.

1899 LORD, JOHN R., M.B., C.M., London County Asylum, Bexley, Kent.

1897 LOWENTHAL, MAX, M.D., M.R.C.P., 126, Princes' Road, Liverpool.

1898 MACCORMAC, JOHN M'GEE, M.D., L.R.C.P., L.R.C.S.Edin., 31, Victoria Place, Belfast.

,, MCALDOWIE, ALEXANDER M., M.D., 6, Brook Street, Stoke-on-Trent.

1891 MACDONALD, PETER WILLIAM, M.D., C.M., The County Asylum, Dorchester, Dorset.

,, MACKENZIE, HECTOR WILLIAM GAVIN, M.D., F.R.C.P., 59, Welbeck Street, W.

O.M. MACKENZIE, STEPHEN, M.D., F.R.C.P., 18, Cavendish Square, W. (C., 1896-98).

1896 MACKINTOSH, ASHLEY W., M.A., M.D., Wellington House, Alford Place, Aberdeen.

1899 MACLULICH, PEERS, M.D., B.Ch., Joint Counties Asylum, Carmarthen.

Elected

1889 MACPHAIL, S. R., M.D., Borough Asylum, Rowditch, Derby.

O.M. MAGUIRE, ROBERT, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)

,, MANN, J. DIXON, M.D., F.R.C.P., 16, St. John's Street, Manchester.

1894 MARTIN, SIDNEY, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.

1891 MAY, W. PAGE, M.D., M.R.C.P., Helouan, Cairo, Egypt.

1890 MENZIES, WILLIAM FRANCIS, M.D., B.Sc., M.R.C.P., Cheddleton Asylum, Leek, Staffordshire.

O.M. MERCIER, C., M.B., M.R.C.P., Flower House, Southend, Catford, S.E. (C., 1899-1900.)

1897 MEYER, ADOLF, M.D., Clark University, Worcester, Mass., U.S.A.

O.M. MICKLE, W. JULIUS, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (V.P., 1900; C., 1895-97.)

1895 MOORE, NORMAN, M.D., F.R.C.P., 94, Gloucester Place, W.

O.M. MORRIS, MALCOLM, F.R.C.S.Ed., 8, Harley Street, W.

1888 MOTT, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894-98; C., 1899-1900.)

1899 MOULD, GILBERT E., M.R.C.S., L.R.C.P., Dyffryn Aled, Abergele.

1894 MURRAY, GEORGE R., M.A., M.D., F.R.C.P., 11, Ellison Place, Newcastle-on-Tyne.

1889 MURRAY, H. MONTAGUE, M.D., F.R.C.P., 27, Saville Row, W.

O.M. NETTLESHIP, EDWARD, F.R.C.S., 5, Wimpole Street, W.

,, NICOLSON, DAVID, C.B., M.D., M.R.C.P.Ed., Elmhyrst, Guildford, Surrey.

1888 NIERMEYER, J. H. H., M.D., Amsterdam.

,, NORMAN, CONOLLY, F.R.C.P.I., F.R.C.S.I., St. Dymphna's, N. Circular Road, Dublin.

1887 OGILVIE, GEORGE, M.B., M.R.C.P., 22, Welbeck Street, W.

,, OGILVIE, LESLIE, M.B., M.R.C.P., 46, Welbeck Street, W.

,, OLIVER, THOMAS, M.D., F.R.C.P., 7, Ellison Place, Newcastle-on-Tyne.

O.M. ORANGE, WM., C.B., M.D., F.R.C.P., The Bryn, Godalming, Surrey.

,, ORD, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.)

,, ORMEROD, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97.)

,, ORR, DAVID, M.B., County Asylum, Prestwich, Manchester.

,, PAGE, HERBERT WILLIAM, M.A., F.R.C.S., 146, Harley Street, W. (V.P., 1900, C., 1891-92.)

1895 PARKINSON, JOHN PORTER, M.D.Lond., M.R.C.P., 57, Wimpole Street, W.

1896 PATERSON, DONALD ROSE, M.D., M.R.C.P., 18, Windsor Place, Cardiff.

1895 PINDER, GEORGE, M.D., B.C., Hazeldene, Farncombe Road, Worthing.

1899 PIPER, FRANCIS P., M.B., M.R.C.S., L.R.C.P., Claybury Asylum, Woodford, Essex.

Elected

1888 PITT, G. N., M.D., F.R.C.P., 15, Portland Place, W.

O.M. POORE, G. VIVIAN, M.D., F.R.C.P., 32, Wimpole Street, W. (C. 1898-99.)

,, PURVES, W. LAIDLAW, M.D., 20, Stratford Place, W.

1899 RAMBAUT, DANIEL F., M.D., B.Ch., Richmond District Asylum, Dublin.

1894 RANSOM, WILLIAM B., M.D., F.R.C.P., The Pavement, Nottingham.

1890 RAYNER, HENRY, M.D., M.R.C.P.Ed., 16, Queen Anne Street, W.

O.M. REID, E. W., M.B., F.R.S., University College, Dundee.

1897 REISSMANN, CHARLES H., B.A., B.Sc., Saxony Villas, Oppidans Road, Primrose Hill.

1893 RENNIE, GEORGE E., M.D., M.R.C.P., College Street, Sidney.

1895 REYNOLDS, ERNEST S., M.D., F.R.C.P., 23, St. John Street, Manchester.

1892 RICHARDS, JOSEPH PEEKE, M.R.C.S., 6, Freeland Road, Ealing, W.

1891 RIVERS, W. H. R., M.A., M.D., F.R.C.P., St. John's College, Cambridge. (C., 1897-98.)

1898 ROBERTSON, WILLIAM FORD, M.D., 12, Bristo Place, Edinburgh.

1892 ROWE, EDMUND LEWIS, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

,, RUFFER, MARC ARMAND, M.D., Cairo.

1889 RUSSELL, J. S. RISIEN, M.D., F.R.C.P., 4, Queen Anne Street, W. (Sec., 1896-1900.)

1899 RUSSELL, J. W., M.D., M.R.C.P., 72, Newhall Street, Birmingham.

O.M. SANDERSON, Sir J. BURDON, M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.

,, SAVAGE, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres., 1897; V.P., 1895; C., 1886-90, 1898.)

1888 SAVILL, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. SCHÄFER, E. A., F.R.S., University of Edinburgh. (Pres., 1893; V.P., 1892; C. 1886-90.)

,, SEMON, Sir FELIX, M.D., F.R.C.P., 39, Wimpole Street, W. (C., 1899-1900.)

1898 SEWARD, WM. JOSEPH, M.B., Colney Hatch Asylum, N.

O.M. SHARKEY, S. J., M.D., F.R.C.P., 22, Harley Street, W. (C., 1889-92.)

,, SHERRINGTON, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C. 1892-95.)

1894 SHUTTLEWORTH, GEORGE E., M.D., Ancaster House, Richmond.

1898 SIMPSON, FRANCIS O., L.R.C.P., M.R.C.S., Rainhill Asylum, Liverpool.

1895 SMITH, F. J., M.D., M.R.C.P., 138, Harley Street, W.

1887 SMITH, R. PERCY, M.D., F.R.C.P., 36, Queen Anne Street, W.

1895 SMITH, TELFORD, M.D., B.S., 54, South Hill Park, Bromley, Kent.

,, SPICER, W. T. HOLMES, M.D., F.R.C.S., 47, Welbeck Street, W.

,, STANSFIELD, THOS. E. K., M.B., London County Asylum, Bexley, Kent.

Elected

1895 STANLEY, DOUGLAS, M.D., 9, Easy Row, Birmingham.

1892 STARLING, ERNEST HENRY, M.D., F.R.C.P., F.R.S., 8, Park Square, N.W.

1897 STEWART, PURVES, M.A., M.D., M.R.C.P., 133, Harley Street, W.

,, STIRLING, WM., M.D., D.Sc., Owens College, Manchester.

1897 STODDART, WM. H., M.D., B.S., M.R.C.P., Bethlem Royal Hospital, Lambeth, S.E.

1895 STOUT, G. F., M.A., St. John's College, Cambridge.

1887 SUCKLING, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.

1889 SUTHERLAND, HENRY, M.D., M.R.C.P., 21, New Cavendish Street, Portland Place, W.

1888 SYERS, HENRY W., M.D., M.R.C.P., 75, Wimpole Street, W.

1898 TAYLOR, F. R. P., M.D., B.S., Darenth Asylums and Schools, Dartford, Kent.

1891 TAYLOR, JAMES, M.D., F.R.C.P., 49, Welbeck Street, W. (C. 1900.)

1898 THOMSON, H. CAMPBELL, M.D., M.R.C.P., 34, Queen Anne Street, W.

1889 THORBURN, WM., F.R.C.S., The Limes, Nelson Street, Manchester.

1892 TITCHENER, E. B., B.A., Ph.D., 72, Heustis Street, Ithaca, N.Y.

O.M. TOOTH, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896-99.)

1892 TREVELYAN, E. F., M.D., B.Sc., M.R.C.P., 40, Park Square, Leeds.

1892 TUCKEY, CHARLES LLOYD, M.D., C.M., 88, Park Street, Grosvenor Square, W.

1892 TUKE, Sir J. BATTY, M.D., F.R.C.P.Ed., 20, Charlotte Sq., Edinburgh.

1891 TUKE, THOMAS SEYMOUR, M.B., M.R.C.S., Chiswick House, Chiswick.

,, TURNER, WILLIAM ALDREN, M.D., F.R.C.P., 13, Queen Anne Street, W.

1898 TURNEY, HORACE G., M.D., M.Ch., F.R.C.P., 68, Portland Place, W.

O.M. TWEEDY, JOHN, F.R.C.S., 100, Harley Street, W.

1888 VOORTHUS, J. A., M.D., Apeldoorn, 542, Loulaan, Netherlands.

1895 WADE, ARTHUR L., M.D., Somerset and Bath Asylum, Wells.

1894 WALKER, A. STODART, M.B., 30, Walker Street, Edinburgh.

O.M. WALLER, AUGUSTUS, M.D., F.R.S., 16, Grove End Road, N.W. (Pres., 1900; V.P., 1899; C., 1894-97.)

,, WARD, J., D.Sc., Trinity College, Cambridge.

,, WARNER, FRANCIS, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1892 WASHBOURN, J. W., M.D., F.R.C.P., 6, Cavendish Place, W.

O.M. WATTEVILLE, A. DE, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896-1900; Sec., 1886-89; Editor of *Brain*.)

1897 WEBER, F. PARKES, M.D., F.R.C.P., 19, Harley Street, W.

,, WEST, SAMUEL, M.D., F.R.C.P., 15, Wimpole Street, W.

Elected

1897 WHITE, ERNEST W., M.B., M.R.C.P., City of London Asylum, Dartford.

O.M. WHITE, W. HALE, M.D., F.R.C.P., 65, Harley Street, W. (C., 1897-1900).

1894 WHITING, ARTHUR J., M.D., M.R.C.P., 142, Harley Street, W.

1889 WIGLESWORTH, JOSEPH, M.D., F.R.C.P., County Asylum, Rainhill, Lancashire.

1894 WILLIAMSON, RICHARD T., M.D., M.R.C.P., 294, Oxford Road, Manchester.

1896 WILSON, ALBERT, M.D., M.R.C.P., 33, Fairlop Road, Leytonstone.

,, WOOD, GUY M., M.B., M.R.C.P., 9, Endsleigh Street, W.C.

1889 WOOD, T. OUTTERSON, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.

1892 WOODHEAD, G. SIMS, M.D., F.R.C.P.Ed., Pathological Laboratory, Cambridge.

1899 WRIGHT, HAMILTON, M.D., Claybury Hall, Woodford Bridge, Essex.

1898 YOUNG, ROBERT ARTHUR, M.D., B.Sc., M.R.C.P., 41, Wimpole St., W.